

ANDERS

PRACTICE

OF MEDICINE



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# A TEXT-BOOK

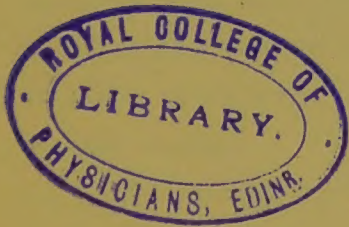
OF THE

# PRACTICE OF MEDICINE

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## P R E F A C E.

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THIS work is meant to introduce the student to the present state of our knowledge of the practice of medicine in general and of the diagnosis, differential diagnosis, and treatment of disease in particular. The historic development of the subjects treated has been either briefly given or intendedly omitted, since this scarcely falls within the scope of a practical treatise on medicine. Although the book as a whole is submitted to the critical judgment of a learned profession, it may be pardonable to emphasize, provisionally, a few features pertaining to the mode of treating the separate subjects, or the arrangement of the material under the latter—to indicate some of the more salient lineaments, so to speak, in the general design. Since in medical schools it is taught from a separate chair, the pathology (special) of the individual affections has almost invariably been taken up before the etiology; from this point the student will find the story of each affection a continuous one. The practitioner, however, must ever aim to associate the clinical symptoms with the morbid lesions.

Under special etiology the bacteriology has been prominently mentioned, since we owe to it the rapid progress that is being made in the study of the causation of disease.

The differential diagnosis has in many instances been tabulated—an ear-mark that I confidently believe will be found especially helpful. It may be stated that not less than fifty-six diagnostic tables are scattered throughout the work, and that by far the greater number of these are my own.

Such formulæ have been introduced into the text, and only such, as a more or less extended experience has shown to be possessed of real therapeutic importance. Whilst these, and all additional points relating to the treatment of the single affections, may serve as guides, particularly to the beginner, I fully appreciate how often the practising physician is



placed in a position in which he is compelled to form a therapeutic judgment for himself. Whenever the dosage is stated, the metric equivalent is placed in parentheses, the number of grams being stated in round numbers (3j—4.0; 3j—32.0) in order to render it of greater practical value. In all instances, however, in which this would involve an important difference in quantity the exact decimal figures are given. A considerable variation from the usual classification of diseases may be observed, but this is accounted for in the text wherever it occurs.

Preference has been given to the modern orthography and terminology, not only because it is more euphonious, but also because of its adoption by the standard lexicographers.

I have gleaned without stint from medical literature with a view to bringing the book up to date, and if I have failed to give full credit in every instance, my grateful acknowledgments are here due and are cheerfully made. The chief results of my personal experience and observation, extending over a period of two decades, and derived from both hospital and private practice, will also be found upon these pages.

I wish to thank Prof. W. C. Hollopeter, who has written some of the articles upon the diseases of children, as measles, chicken-pox, mumps, whooping-cough, and the acute diarrheas, and who has kindly aided in the preparation of those upon diphtheria and scarlatina.

My cordial thanks are due also to Dr. C. L. Furbush for kind aid in preparing some of the illustrations, to Doctors Robert N. Willson, Howard S. Anders, and Geo. W. Pfromm for valuable assistance while the work was passing through the press, and to Dr. A. M. Davis for preparing the index.

JAMES M. ANDERS.

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## PART I.

# INFECTIOUS DISEASES.

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### TYPHOID FEVER.

(*Enteric Fever; Abdominal Typhus; Ileo-typhoid; Nerven Fieber.*)

**Definition.**—An acute infectious disease of which the definitive cause is the specific bacillus of Eberth. It is characterized, pathologically, by hyperplasia and sloughing of Peyer's patches; and clinically by its slow, insidious onset, peculiar temperature-curve, swelling of the spleen, rose-colored spots, diarrhea, tympanites, and a liability to certain complications (intestinal hemorrhage, peritonitis, etc.). The disease has an average duration of from three to four weeks.

**History.**—Although known beyond the reach of tradition, typhoid fever was clearly distinguished from typhus at a comparatively recent date. Louis of Paris in 1829 proposed the term *typhoïde*, but it remained for Gerhard of Philadelphia to discriminate typhoid from typhus fever as the result of his own precise clinical observations. His account of the disease was ably corroborated by the writings of E. Hale and James Jackson, Sr. (1838, 1839). Later, Shattuck of Boston and Jenner of London made important contributions to the subject. Shattuck's experiments on typhus and typhoid fevers at the London Fever Hospital in England, and Alfred Stillé's studies of the former affection in Dublin and Naples, and of the latter in Paris, increased greatly our knowledge of these diseases. As a result of the labors of the above-mentioned American authors the true nature and identity of typhoid fever were appreciated in America at an earlier day than in either France or England.

Briefly, the decade from 1840 to 1850 witnessed, on the one hand, the overthrow of erroneous notions concerning the similarity of typhoid and typhus fevers, and, on the other, the establishment of their points of dissimilarity.

**Pathology.**—The lesions produced by typhoid fever may conveniently be divided into two groups: (1) Primary lesions, due to the direct effect of the special bacillus upon the lymph-follicles of the intestines, the mesenteric and other lymph-glands, and the spleen. (2) Secondary lesions, due chiefly to the long-continued fever and to secondary infection, for the occurrence of which the essential lesions of typhoid fever furnish the golden opportunity.

(1) The primary morbid changes in the Peyer's patches and solitary glands of the intestines are divided, usually, into four stages:

(a) **The Stage of Infiltration.**—The lymph-follicles become engorged



(hyperplasia), particularly Peyer's glands in the ileum and near to the valve, and, to a lesser extent, in the lower part of the jejunum. Frequently, the solitary glands in the small intestines, the colon, and rarely the rectum, become similarly infiltrated. In about 33 per cent. of the cases the chief morbid lesions are confined to the large intestines. In mild cases a few Peyer's patches in the lower part of the ileum are alone the seat of infiltration and subsequent changes. The follicles are grayish-white in color, and may project—particularly the patches of Peyer—from 3 to 5 mm. or more. Rarely, the solitary glands, which vary in size from a mustard-seed to a large pea, become very prominent and show a bold attempt at pedunculation.

The *histologic changes* at first consist in a marked dilatation of the capillary blood-vessels, which later are more or less compressed (as a consequence of cell-infiltration), giving to the follicles their whitish, anemic appearance. The cellular elements partake of the nature of lymph-corpuscles. Some of these cells are larger and are epithelioid in character, with ten or more nuclei. The mucosa and muscularis adjacent to the glandular structures may be similarly infiltrated.

From the eighth to the tenth day the stage of infiltration terminates either in resolution or in necrosis and sloughing. The infiltrated cells may undergo granular or fatty degeneration, followed by absorption. This process—resolution—during its progress produces pitting of the swollen follicles. In consequence of these minute points of necrosis the plaques now present a characteristic reticulated appearance (plaques à surface réticulée). When resolution occurs, accompanied by destruction of the follicles, small hemorrhages may take place into the glandular structure. These hemorrhages may occasion pigmentary deposits in the follicular depressions, giving rise to the so-called "shaven-beard" appearance. Resolution, however, terminates the stage of medullary infiltration with relative infrequency. Far more frequently the hyperplasia of the lymph-follicles ends in

(b) **Necrosis or Sloughing.**—In all save the milder grades of cell-infiltration the hyperplasia of the lymphatic tissue cannot subside before necrosis occurs. The latter process results partly from compression and choking of the blood-vessels by the cell-infiltration, and partly from the direct action of the typhoid bacillus, leading to so-called anemic necrosis. Thus, necrotic crusts (sloughs) are formed, which are gradually separated and cast off. While not all of the glands of Peyer which are the seat of cellular infiltration undergo subsequent necrosis, as a rule those situated in the lower portion of the ileum do, and show the process in its completest development. The depth to which the necrosis extends is quite variable. It may involve only the most superficial layers of the mucosa, or it may extend in depth till it reaches, or even perforates, the outer or serous coat; but usually this work of destruction does not dip below the submucosa or muscularis. The necrosed portions become detached—a process that proceeds from the periphery toward the center—leaving behind the typhoid ulcer. The stage of necrosis and sloughing begins between the eighth and tenth days, and ends on or about the twenty-first day.

(c) **Stage of Ulceration.**—The size and shape of the ulcers correspond exactly to the necrosed areas in these respects. A single gland

of Peyer generally presents several ulcers of irregular outline separated by strips of mucous membrane. Rarely, the entire plaque is implicated, in which case a large oval ulcer is the result, and at the lower end of the ileum the ulcers often coalesce until they almost encircle the bowel. The ulcers of the solitary glands assume a rounded form. The character of the floor of the ulcer will vary with the character of the intestinal coat which forms its base, though usually it is clean and smooth. The edges are usually irregular, engorged, soft, and frequently overhanging. In the lower segment of the ileum ulcers may be numerous, whilst in other portions of the gut Peyer's plaques may be merely hyperemic. In about 25 per cent. of the cases the typhoid ulcers are found in the large intestines—*i. e.* in the cecum and colon. Perforation of the large bowel is exceedingly rare. Exceptionally, the appendix is the seat of ulcer, Osler having dissected a case that died three months after an attack of typhoid fever, in which he found a localized abscess due to perforation of the appendix.

*Hemorrhage* usually results from erosion of a vessel—an accident which is occasioned by the separation of the sloughs—but small bleedings may take place from the swollen, hyperemic edges of an ulcer. *Perforation* of the bowel occurs in a small percentage of cases (about 6 per cent.). In the majority of instances it is attributed to a perforative necrosis; hence it is that the sloughs are usually found attached to the orifice. Perforation may also occur after the separation of the necrosed portions during the stage of ulceration. The perforations may be multiple, though they are usually single and rarely exceeding two in number. The small, deep ulcers are more apt to lead to complete perforation than larger ones, and the site of the orifice is usually somewhere in the course of the lower third of the ileum. The lesions of peritonitis are invariably present, and during the stages of necrosis and ulceration a catarrhal state of the mucosa of the small and large intestines exists. The diarrhea which usually accompanies this affection is to be ascribed chiefly to the catarrhal state of the large bowel.

(*d*) **Healing** follows promptly upon the formation of the ulcer. At first a granular tissue covers its floor. The mucous membrane is replaced, including the glandular elements and epithelial layer, and, as in the stage of necrosis and sloughing, so the healing process advances inward from the border of the ulcer. Indeed, it is this process that dislodges the necrotic crust. Occasionally, ulcers are seen extending in one direction while healing in another. The cicatrix formed by the healing of an ulcer presents a smooth and often pigmented surface.

The stages thus far described do not, strictly speaking, follow one another, since two or more may be illustrated at once by a group of ulcers occupying the same section of the intestine. Again, when death occurs during a relapse fresh ulcers are observed by the side of others that are partially healed.

*The Mesenteric Glands.*—Changes in the mesenteric glands occur simultaneously with those in the intestines, and those situated opposite to the lower third of the ileum, the portion of the bowel showing the most extensive ulceration, are most profoundly involved. Hyperemia, and later swelling due to cell-infiltration, are among the earliest



changes, and correspond with the lesions noted in the intestines (*vide supra*). The mesenteric glands exhibit great variations in size, ranging, as they do, from that of a pea to a hen's egg. Their color-appearance is a grayish-red. Resolution occurs quite commonly, but, if it does not take place, then necrosis of the central portion (due, most probably, to the same causes that produce necrosis of the intestinal lymph-follicles) occurs, and suppuration has been observed in some cases. Still other glands become hyperemic and swollen (retroperitoneal, bronchial, etc.); but these almost invariably tend toward resolution.

*The Spleen.*—With rare exceptions the spleen becomes enlarged in typhoid fever. At first hyperemic, the tissue then grows soft and granular, and at times is almost diffuent on section. Infarction is not a rare occurrence and may lead to suppuration. In some few cases, either spontaneously or as the result of injury, a rupture of the organ may occur, and the records of 2000 post-mortems at the Munich Pathologic Institute furnish 5 instances of this nature.

(2) **Secondary Lesions due chiefly to the Continued Fever and to Secondary Infections.**—The lesions in other organs are of subsidiary importance, and are, for the most part, secondary in nature, though we cannot, in the present state of our knowledge, draw a sharp line of distinction between these lesions and those that are primary. While the pathologic changes above described are chiefly due to the direct action of the specific bacillus of typhoid, yet a few of them are ascribed to secondary infection, and, at all events, they do not belong peculiarly to this disease—*e. g.* suppurative processes, etc. Further, in connection with the clinical history of the affection I shall point out that in a small percentage of cases the initial specific lesions may be localized, either in the throat or in the lungs or kidneys. It is to be emphasized, therefore, that whilst the essential pathologic processes of the disease have been described above, a classification of the lesions into primary and secondary, based upon the involvement of particular organs, can only be approximately correct.

The *liver* early becomes hyperemic, and later is softer and paler than is natural. Handford has described necrotic areas, and Wagner minute lymphomata. Infarction and abscess occur in rare instances. The mucosa of the *gall-bladder* may show catarrhal inflammation, and in very rare instances a croupous, diphtheritic, or ulcerative inflammation of this organ may occur. The bile is thinner and paler than the normal.

The microscope reveals parenchymatous and granular degeneration. The cells contain an abundance of fat, whilst their nuclei have lost, in great part, their outline.

The *kidneys*, like the liver, exhibit parenchymatous degeneration. They are somewhat pale-looking, are cloudy on section, and slightly swollen, and under the microscope granular and fatty degeneration of the epithelial cells of the convoluted tubules is observed. More rarely the lesions are those of *acute hemorrhagic nephritis*. Small areas of round-cell infiltration may develop late in the course of typhoid, and these may present an appearance similar to lymphomata or may undergo softening and suppuration, giving rise to miliary abscesses. The mu-

cous membrane of the pelvis of the kidney is not infrequently the seat of a mild grade of catarrh, and, rarely, of diphtheritic inflammation. Vesical catarrh is still more common, and the bladder may also be the seat of diphtheritic inflammation. Rarely orchitis is encountered. On making cultures from sections of the kidneys not a few observers have been able to demonstrate the specific bacillus of typhoid, particularly in the softened areas.

In the *lungs* are found morbid lesions in nearly all cases of typhoid fever, and belonging to the essential pathologic processes is bronchitis, due to a congested and catarrhal state of the bronchial mucous membrane. The lesions of lobular pneumonia present a complicating condition in many instances; those of lobar pneumonia also may be present, though less commonly. The so-called *hypostatic congestion* is often found, but is, I think, less frequent than is supposed by many authors. *Embotic infarctions*, having their origin in thrombi occupying the right side of the heart, are sometimes present. Gangrene may also occur.

*Pleurisy* is sometimes, though rarely, met with. It is most frequently of the plastic variety, although empyema occurred in nearly 2 per cent. of the Munich cases.

The *larynx* and the *pharynx* may manifest changes. Ulcers have been observed on the epiglottis and posterior wall of the larynx, and I have more than once seen them on the pharynx. When situated in the larynx they may extend in depth till they reach the perichondrium, causing perichondritis, with or without edema of the larynx as an associated lesion. Typhoid bacilli have been found in the ulcers (Eichhorst). Catarrhal, or even croupous, pharyngitis may occur, and a swelling of the follicles of the pharynx and base of the tongue is to be noticed in many cases. True aphthous changes, affecting the mouth and pharynx, may be present as a secondary event. The *mucosa of the stomach* is sometimes congested, and may even ulcerate, although this is very rarely seen.

*Peritonitis* is always found in fatal cases in which the bowel has been perforated. The condition is a general one, save in the rare instances mentioned below, and there is usually much fibrino-purulent effusion present. Diffuse peritonitis may be present without perforation, and results sometimes from a localization of the typhoid poison in the peritoneum, from rupture of suppurating mesenteric glands, but more frequently, I think, from direct extension of intestinal inflammation to the peritoneum.

The *heart* may be the seat of morbid changes. Acute endocarditis may be a very rare complication, while pericarditis occurs relatively more often—viz. in 14 of the Munich post-mortems before mentioned. Myocarditis is a not uncommon event, the cardiac muscle exhibiting parenchymatous and, less commonly, hyaline, degeneration, and the latter change sometimes leads to sudden rupture of the muscular fibers, with a fatal result (myocardite ségmentaire). It is, however, a significant fact that in the majority of instances, even of the severest type, the cell-fibers may show slight, if any, noticeable change. Out of 48 cases, 16 showed granular or fatty degeneration, and 3 a proliferative endarteritis in the small vessels (Dewevre).

The *arteries* have, in a number of instances, been found to be the



seat of two forms of arteritis (Barié): (a) Acute obliterating arteritis, and (b) Partial arteritis. These conditions may affect the smaller vessels, particularly those of the heart, but they occur most commonly in the arteries of the lower extremities. Thrombi are found in the right chambers of the heart and in the veins—most frequently in the femoral, and less often in the cerebral sinuses.

The *voluntary muscles* undergo parenchymatous and, occasionally, a hyaline, change, though this is not a feature peculiar to typhoid fever. The latter form of degeneration does not affect the whole muscle, only certain fibers being involved, and as a rule the recti abdominis, the diaphragm, the adductors of the thigh, and the pectorals are the seats of the lesion. The parts affected are pale and possess a grayish, waxy luster. Histologically, the process implies the transformation of the muscular fibers, and especially the cement substance, into a homogeneous, pliable mass. Regeneration of the fibers thus destroyed occurs during convalescence.

The *nervous system* presents no gross lesions, if we except meningitis, the latter occurring as a complication; but it is exceedingly rare, having been present in only 11 of the 2000 Munich cases. In a few instances large cerebral hemorrhages have been met with, but these are apparently coincidental, while capillary hemorrhages into the cortex may be numerous. Meningeal hemorrhages may also occur. Slight edema of the cerebral cortex has been noted. The peripheral nerves are, not infrequently, the seat of parenchymatous change, with or without local neuritis, and the ganglia of the trunks of the vagi exhibit an inflammatory change, which Levin feels is the cause of certain symptoms and conditions, such as laryngitis, pharyngitis, pharyngolysis, arrhythmia, etc.

The *blood* shows few important alterations. The red blood-corpuscles are relatively increased during the febrile period and markedly diminished during convalescence, but the great loss of water during the former period and a reabsorption during the latter will explain these interesting facts (Henry). Leukocytosis is absent, and there is often an actual decrease in the number of leukocytes. The mononuclear forms are more numerous than in health.

**Etiology.—Bacteriology.**—The bacterium which is the specific cause of typhoid fever was discovered by Eberth, whose researches were later confirmed by the careful investigations of Gaffky. It is a short, thick bacillus, about three times as long as it is broad, with rounded ends (Fig. 1). It is motile, due to the presence of cilia on both sides, and when stained exhibits vacuolations that have been mistaken for spores. It is easily stained with all the anilin dyes. It has been found in the intestinal tract, in the lymph-glands, contents of the intestine, spleen, liver, and blood; in fact, it has been found in nearly all the organs of the body. Upon gelatin plates it develops in grayish translucent colonies with irregular borders and ridged surfaces. Upon agar the growth is not characteristic; upon the potato, especially if it has been rendered slightly acid, it forms a perfectly transparent growth that is only evident as a slight apparent increase of moisture upon the surface, and as offering a greater resistance to the point of the needle when scraped across it. It neither coagulates milk, liquefies gelatin,

nor produces indol. The organism never forms spores. Gaffky described the formation of spores, and his observation was afterwards confirmed by Chantemesse and Widal, but Buchner and Pfeil subsequently showed that the suspicious bodies were merely conglomerations of protoplasm that had undergone a plasmolytic process. Moreover, the bacillus has no more powers of resistance than the ordinary bacteria. Inoculated into lower animals, it frequently causes fatal results without producing the lesions characteristic of typhoid in human beings, although occasionally typical typhoid ulcers have been found. The susceptibility of lower animals, though normally slight, can be increased by preliminary injections of saprophytic bacteria, this result having been observed by Alessi when he exposed animals to the gases produced by putrefying matters. The poison is probably a toxin, and it is quite possible to render the lower animals immune to disease. Usually in making a bacteriologic diagnosis the typhoid bacillus is to be separated from those organisms that morphologically resemble it and present almost identical characteristics upon various culture-media. Particularly is this true of the bacterium coli commune, which differs, though not invariably, in the fact that it produces fermentation of the saccharine media, or forms indol in peptone bouillon, and coagulates milk, with the production of an acid reaction. Elsner has proposed cultivation of the fecal organisms upon acid glycerin potato to which 1 per cent. of potassium iodid has been added. At the end of twenty-four hours the typhoid bacillus has formed minute, grayish-white points upon the culture-media, while the bacterium coli commune has formed luxuriant colonies. At the end of forty-eight hours the typhoid bacilli appear in clear shining drop-like and finely granular colonies, and the bacterium coli commune in much coarser granular colonies that are brownish in color.

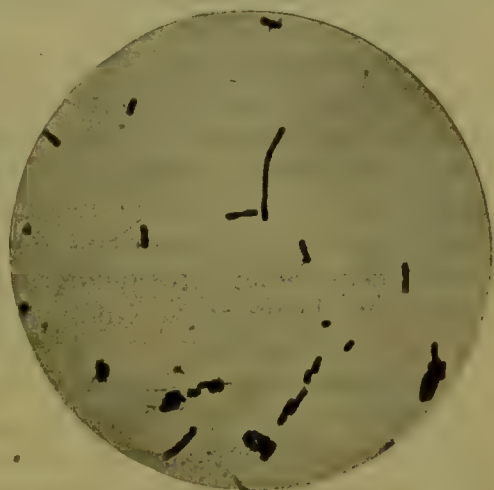


FIG. 1.—Typhoid bacilli with flagella;  $\times 1000$ .

Of late this method has been found more or less inaccurate, and Elsner himself admits that it is untrustworthy.

The real poison of typhoid fever must, therefore, be a chemical substance secreted by the bacillus—*typho-toxin*—and Brieger has extracted the latter agent, finding that it produces the fever, nervous symptoms, and the other manifestations characteristic of the affection. Most authors agree that the bacilli cannot maintain a permanent existence outside the human body. From time to time, however, the conditions indispensable to the growth and development of the typhoid germs prevail, and corresponding with such periods of time more or less extensive epidemic outbreaks of the disease may occur. It is known that the typhoid bacilli may retain their vitality from seven to fourteen days in water, disappearing from the same on account of the presence of saprophytes; but an epidemic or an endemic of typhoid fever implies persistent con-



tamination of the drinking-water. Multiplication of the bacilli may take place in water, in milk (very rapidly), and in the soil (where they preserve their vitality under favorable conditions for months). Freezing does not kill them, as they may live in ice for several months (Prudden). They have been discovered in infected water, but are thoroughly destroyed by boiling.

**Predisposing Causes.**—Typhoid fever is particularly prevalent in temperate latitudes and in every quarter of the globe. Among the influences predisposing to the disease are—

(a) **Geographic Location.**—In temperate zones it prevails constantly and to a greater or less extent. But, though the disease occurs most frequently in temperate climates, it cannot safely be inferred that climate *per se* exerts any marked influence over its appearance. Thus it has been shown in recent times to be comparatively common in the tropics as well as in many cold latitudes (Iceland, Norway, etc.).

(b) **Seasons** exert a decided influence upon the frequency of the occurrence of typhoid. According to the statistics of Murchison, Bartlett, Osler, Hirsh, and others, the time of greatest liability to typhoid fever is during the late summer and the early autumn, the months in which cases are most frequent being August, September, and October. The remaining summer and autumn months yield a relatively larger number of cases than the winter and spring: again, in winter more cases are met with than in the spring, which furnishes fewer cases than any other season of the year. After dry summers typhoid fever is especially apt to be prevalent, and, according to Baumgarten, a relatively large amount of dust in the atmosphere may disseminate the typhoid germs.

(c) **Condition of the "Ground Soil."**—Pettenkofer and his disciples contend that when the standing water in the soil reaches a high level fewer cases occur, and when it falls to a low level or below the mean height the cases become more numerous. This dictum, however, has not as yet been conclusively proven with reference to many localities. Whilst the condition of the soil as to moisture, etc. cannot explain all of the peculiarities noticeable in the behavior and distribution of the disease, certain characteristics of the soil may furnish the conditions essential to the growth, development, and multiplication of the typhoid bacillus, and it is certain that epidemics of typhoid fever occur repeatedly without regard to the condition of the ground-water.

(d) **Age.**—Typhoid fever may occur at any age. It is, however, especially frequent among young, robust individuals between the ages of fifteen and thirty years. Later in life it becomes progressively less frequent, though cases have occurred at or beyond the seventieth year. Young children are not exempt, and cases among them are of rather frequent occurrence, if we except those under one year of age.

(e) **Sex** probably does not affect the degree of liability in typhoid fever.

(f) **Individual Predisposition.**—This may be *acquired* or *inherited*. An instance of *acquired* predisposition is to be noted in the great susceptibility which exists among persons who have recently moved from rural districts to cities. Thus, Louis found "that of 129 cases, 73 had not resided in Paris over ten months, and 102 not over twenty months."

To account for this fact we have the influence of a change both of sanitary surroundings and of the habits of life. That the predisposition to this affection may also be *inherited* I have no doubt. We must assume in these cases the existence of certain peculiarities of the family soil to account for the heightened receptivity that is so often observed in and throughout successive generations. Most persons, however, and perhaps most families, seem to enjoy natural immunity from the affection.

(g) **Intestinal Catarrh.**—It is quite probable that a catarrhal state of the intestinal mucosa produces local predisposition to infection with the typhoid poison. I have observed cases of influenza, with marked catarrh of the gastro-intestinal tract, to be followed promptly by the symptoms of typhoid fever (*vide* Influenza). The occurrence of typhoid fever confers an approximate, though not an absolute, immunity against subsequent attacks.

(h) **Nervous Influences.**—Great mental excitement and overwork are among the predisposing nervous causes.

In this connection two questions present themselves for consideration :

(1) What are the methods of conveyance of the poison into the human body?

In the first place, isolated cases and epidemics of typhoid fever are alike to be attributed to antecedent cases of the disease, and this fact presupposes that the bacillus of typhoid leaves the body of the sufferer during the attack. This it does in the stools, which are practically the only primary source of infection and contain the bacilli or their spores in great numbers. From the dejecta the bacilli may be conveyed to well persons by—

(a) **Infected Drinking-water.**—In the vast majority of instances the poison is transmitted from those affected with the disease to those in good health through the drinking-water supply. This has been especially true in those extensive epidemic outbreaks in which the mode of origin has been traced. Wells, storage reservoirs, springs, and rivers may alike become contaminated and cause epidemic prevalence of the disease.

In the spring of 1885 a most instructive, though deplorable, epidemic occurred in Plymouth, Penna., a town of 8000 inhabitants. At first the nature of the affection was not recognized, and before it ceased to appear 1200 persons were affected, with 130 resulting deaths. This epidemic was investigated by Shakespeare and L. H. Taylor, and was found to have arisen from a single case of typhoid occurring in a house on a hill which sloped toward the water-supply of the town. This patient was ill during January, February, and March, while the ground was frozen and covered with snow, upon which the dejecta were thrown by the attendant. On March 25th there was a considerable rainfall, followed by a sudden thaw, and the water, unable to sink in the frozen earth, ran at once through the various surface channels into a brook, which in turn emptied into the reservoir. Coincidentally with the thaw the patient had frequent and copious stools, and, strangely enough, for certain reasons the infected water-supply was at the same time more largely drawn upon than usual. On April 10th other cases of the disease appeared, and careful investigation showed that those citizens who



obtained their water from other sources than the infected reservoir escaped the disease.

(b) **Infected milk** frequently conveys the poison. It may become polluted by water which has been used either to wash the cans or for diluting purposes, or the bacilli may be transferred to milk by the unclean hands of the milker. Numerous instructive epidemics, originating in infected milk, have been reported (Murchison, Ballard, Almquist, and others).

Solid forms of food (salads, celery, fruits, etc.) may be contaminated by infected water or dust or by the fingers of the nurse or the patient. A fly which has alighted on the soiled linen of a typhoid patient in a ward may subsequently contaminate the milk or other food (Osler). H. W. Conn has shown that oysters while being fattened or freshened may become infected, and the careful studies of Foote teach us that the typhoid bacillus will not only retain its vitality in the salt water in which the oysters are fed, but that it will live even longer in the oyster itself.

(c) **Contagion or Direct Transmission.**—This necessitates direct contact with the typhoid stools, and its possibility cannot be denied. It affords a ready explanation for contraction of the disease by nurses who attend to the stools, the bed, and the body-linen of the patient, and by laundresses who are also obliged to handle the soiled clothing, and who are affected with great relative frequency.

(d) **"Ground Soil."**—According to Pettenkofer's view (*vide supra*), the typhoid bacilli are rarely, if ever, transferred directly from the sick to the healthy. On the contrary, he contends that the typhoid poison which leaves the body of an infected person must undergo modification or development in the ground-soil before it is potent to cause the disease in question. These facts are opposed to the view that typhoid fever is transferable by contagion.

(e) **Sewer Gas.**—The typhoid stools frequently find their way into the sewers, and if they have not been thoroughly disinfected may there meet with all the conditions favorable to the growth and propagation of the bacilli. Moreover, if under these circumstances the house-drain be defective, typhoid bacilli may escape into and contaminate the atmosphere of homes, infecting receptive occupants. Alessi's experiments go to show that animals are rendered highly sensitive to the typhoid bacillus by the inhalation of gases of putrefaction. But the recent researches of Bergey and of Abbott seem to show that sewer gas is not decidedly deleterious, and it certainly cannot of itself cause typhoid fever.

(2) Through what channel or channels does the typhoid bacillus gain entrance into the human system?

(a) **Almost invariably the bacilli are swallowed with drinking-water or food, particularly milk.** It must not be forgotten that bacilli when inhaled may find lodgement in the mouth, pharynx, etc., and then be carried along into the stomach with the next food or drink that is ingested. In the stomach they meet with the acid gastric secretions, which often destroy them. They may, however, pass into the intestinal canal, where the alkaline juices of the small intestines furnish every condition necessary for their further growth and development. They



penetrate the mucosa and attack primarily the solitary follicles and Peyer's plaques. Next they invade the mesenteric glands, reaching the circulation, spleen, liver, and other organs a little later.

(b) The possibility that the bacilli may reach the blood-stream through the respiratory organs must be conceded; and hence the added possibility that they may set up initiatory lesions either in the tonsils or lungs, passing thence into the circulation, must also be granted, although cases rarely originate in this manner.

**Clinical History.**—**I. Incubation.**—The average duration of the period of incubation, or the time between the introduction of the poison into the system and the appearance of the first active symptoms, ranges from ten days to three weeks, though it sometimes lasts for a longer, and oftener, I think, for a shorter, time. During this period the patient may experience no deviation from health, but in most cases there are prodromal symptoms, such as languor, loss of appetite, nausea, headache, neuro-muscular pains in the back and limbs, a disinclination to exercise, etc. These symptoms last usually from a few days to a week or more.

**II. General Symptomatology and Course.**—On account of the peculiar temperature-curve in typhoid fever its course falls naturally into three periods—the stage of development; the acme or fastigium (corresponding to the height of the disease); and the stage of decline or defervescence. It is convenient to speak of the various weeks of the affection when referring to these stages. Thus, the first week represents the stage of development, the second and third weeks (in cases of average severity) the fastigium, while the fourth week in the typical form (the third week in mild cases) corresponds to the third or declining stage of the disease.

(a) **Stage of Development.**—The invasion, as a rule, is not sudden, but gradual, the symptoms being chilliness and feverishness, with increase in the severity of the prodromal symptoms. Typhoid fever rarely starts in with a distinct rigor. At or about this time nose-bleed betrays the nature of the disease in a considerable proportion of the cases. The symptoms just described are quickly followed by a prostration sufficiently well marked to compel most patients to take their beds. From this latter event is usually dated the onset of the affection. It is safer, however, to regard the time of occurrence of the above-mentioned symptoms (elevation of temperature, with its attendant discomforts) as the time of onset, since many patients continue in their avocations for days after the appearance of the first symptoms.

With the progress of the initial period the symptoms usually increase in severity with considerable rapidity; the temperature rises day by day, till, at the end of four or five days, the second stage, or fastigium, is reached. Anorexia is complete, thirst is great, headache rather intense, the skin hot and dry to the feel, the tongue coated, the sleep disturbed, and constipation often marked. The patient may complain of fits of chilliness, alternating with flushings of heat, and there is a slight cough with some thoracic oppression. The pulse is quickened (from 90 to 110 per minute) and is full, though rarely, thus early, is it dicrotic.

The *physical signs* are not prominent. The abdomen is often slightly distended and tender; the spleen, on *palpation*, is found to be somewhat swollen.

(b) **Fastigium, or the second stage**, commences usually on the fourth or fifth day of the disease, and lasts, in typical cases, about two weeks. During the first week of the fastigium (the second of the disease) the general symptoms become more marked. The fever remains high (the evening temperature usually reaching  $103^{\circ}$  or  $104^{\circ}$  F. ( $40^{\circ}$  C.), and exhibits the continued type. The pulse is accelerated but not dicrotic. The headache disappears, and mental dulness and slowness are conspicuous, but there may be mild delirium, particularly at night. There is a dry cough and the physical signs indicate more or less extensive bronchitis. The tongue is coated and may become dry, the belly is somewhat swollen and tender, and diarrhea replaces constipation. The spleen is decidedly enlarged, and about the eighth day of the disease a number of roseate spots, which are pathognomonic, appear on the trunk. During the latter part of this week a grave or even fatal condition may be developed as a result of intense nervous or pulmonary symptoms, intestinal hemorrhage, or perforation.

During the second week of the fastigium (the third week of the disease) the marked general symptoms already noted persist in severe types of the affection. The pulse varies from 110 to 130, and the temperature may approach the remittent type. In addition, this period furnishes the most numerous as well as the most untoward complications (lobular pneumonia, hypostatic congestion of the lungs, intestinal hemorrhage, perforation, peritonitis, etc.), and in the absence of serious local complications grave general conditions may be presented.

(c) **Stage of Decline or Defervescence.**—At the end of the second stage, and about the twenty-first day of the disease, in favorable cases the fever begins to decline, and with it the other general and local symptoms gradually disappear. This is followed by true convalescence. In protracted cases, however, the fourth week of the disease may present much the same clinical indications as the third, and these may even be intensified. Frequently an aggravated type of the typhoid state is now superadded, the symptoms being stupor, muttering delirium, subsultus tendinum, a rapid, feeble pulse, a dry, brown tongue, marked diarrhea, greatly swollen belly, and an involuntary discharge of feces and urine. Inflammatory complications may add to the perils of the condition.

In not a few cases the febrile period is prolonged into the fifth, and rarely into the sixth or even the seventh week, and the fever observed when defervescence is retarded presents an irregular type. I have elsewhere reported a case in which it lasted not less than seven weeks.<sup>1</sup> About this time recrudescences and relapses may occur in typical cases. Different epidemics of typhoid fever, however, vary so greatly in their clinical characteristics as to make it impossible to include all cases in any outline of the course of the disease that might be attempted. The above sketch embraces the more or less nearly typical cases.

**III. Chief Clinical Features in Detail.**—(a) **Course of the Fever.**—During the stage of development (the first four or five days) the temperature usually rises in “step-ladder” fashion. The evening exacerbation is on each day from a degree and a half to two degrees higher than on

<sup>1</sup> “A Case of Typhoid Fever; numerous Intestinal Hemorrhages, the Amount of Blood Lost being Seventy-eight and one-half Ounces; and Obstinate Vomiting, with Recovery,” *International Clinics*, vol. i. 5th series, April, 1895, p. 29.



the preceding, and the same is true of the morning remissions. A glance at the temperature-charts (Figs. 2 and 3) will show that the morning remissions touch a level from one-half to one degree lower than the preceding evening registers.

When the fastigium is reached, the evening temperature may be  $103^{\circ}$ ,  $104^{\circ}$ , or  $105^{\circ}$  F. ( $39.4^{\circ}$ – $40.5^{\circ}$  C.), and is usually thus maintained, with slight morning remissions, during the first and sometimes during the second week of this period. More often, during the latter half of the fastigium (the third or fourth week of the disease) the morning fall of temperature becomes decidedly greater. According to my own observation, the height of the fastigium is reached a day or two after its onset or at the end of the first week of the affection. Marked morning remissions are a favorable indication. On the other hand, and contrary to the general rule, the morning temperature may be higher than the evening, forming a somewhat unfavorable symptom. Morning temperatures of  $104^{\circ}$  F. ( $40^{\circ}$  C.) or over are indicative of a serious type. In many instances of mild grade the evening temperature at no time exceeds  $103^{\circ}$  ( $39.4^{\circ}$  C.), but oscillates between  $100\frac{3}{5}^{\circ}$  and  $102\frac{3}{5}^{\circ}$  F. ( $38.1^{\circ}$ – $39.2^{\circ}$  C.). In cases of average intensity the morning remissions touch  $102^{\circ}$ – $102\frac{3}{5}^{\circ}$  F. ( $39.2^{\circ}$  C.), and the evening exacerbations reach  $104$ – $104\frac{3}{5}^{\circ}$  F. ( $40.3^{\circ}$  C.). When the temperature rises above  $105^{\circ}$  F. ( $40.5^{\circ}$  C.) hyperpyrexia exists. Ampugnani made studies of hourly charts from 200 cases of typhoid fever, and found the maximum temperature to occur between three and six o'clock in the afternoon, and the minimum between four and eight o'clock in the morning. The duration of the fastigium exhibits a wide range and is dependent upon a variety of conditions—*e. g.* the degree of mildness or severity of the type, the presence or absence of complications, etc. In cases of a mild character it lasts from a few days to one week; in cases of average severity, from ten days to two weeks; in the severest forms, from two to four weeks.

In typical cases the end of the fastigium marks the beginning of the last stage (that of defervescence), and during this period the temperature falls by *lysis*. Measured by days, it declines by degrees, both the morning and evening temperatures being often one or two degrees lower than on the preceding day. Thus is formed a more or less regular step-like line of descent. To this general rule there are two notable exceptions: From the beginning of the period of defervescence the morning remissions may strike the normal point, while the evening exacerbations become less and less marked, until they also touch the normal. Under these circumstances the temperature-curve resembles somewhat that of the quotidian intermittents, and rarely the tertian type of curve obtains. In comparatively rare instances the morning temperature shows a deeper remission on each successive day, while the evening temperature remains high for several days, when it also declines. This period lasts from one week to ten days—a longer time than in the first instance.

In the severe and protracted forms of typhoid fever there occurs between the second stage (fastigium) and the third stage (defervescence) another, to which Wunderlich has given the name of the “ambiguous period.” This lasts from a few days to a week or more, and is charac-



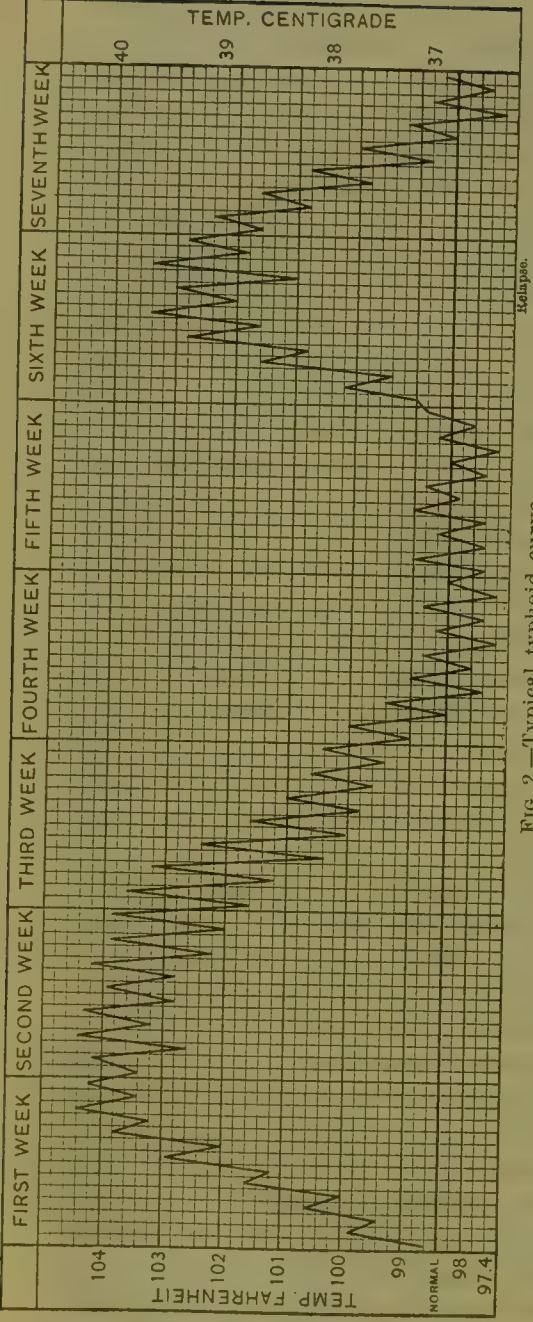


FIG. 2.—Typical typhoid curve.

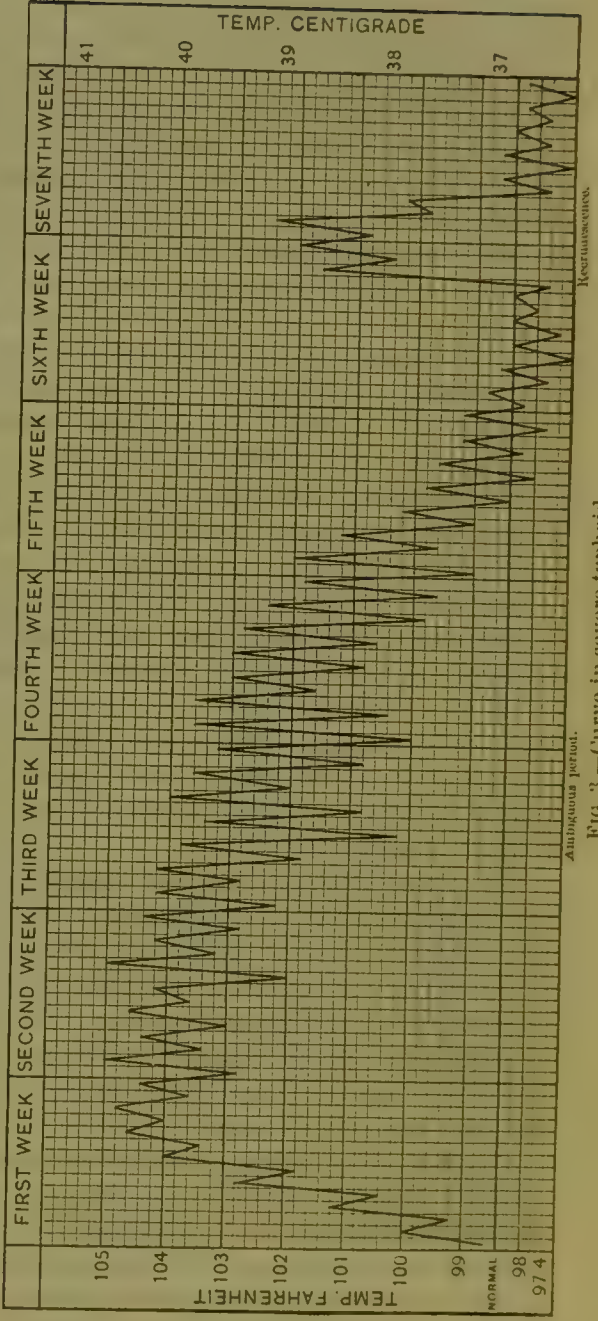


FIG. 3.—Curve in severe typhoid.

terized by a striking diurnal range of temperature, with marked irregularities. It is probable that it is sometimes produced by an auto-infection.

*Abnormal Course of the Fever.*—The pyrexial peculiarities yet to be pointed out are less usual than the foregoing, though of sufficient frequency of occurrence to demand a brief description.

The first stage varies but little from the regular course described above. A sudden elevation of temperature, however, is seen in those rare cases that begin with a severe rigor, and frequently with accompanying pneumonic symptoms. The first stage is but rarely noted by the physician for the reason that the patient does not come under observation at an early period.

As before pointed out, the *fastigium* exhibits the widest variations as to its duration. In the lightest forms it may be practically absent, defervescence setting in upon the first day of the fastigium. There is also a class of cases in which, throughout the greater part of their course, the fever is distinctly intermittent or remittent, and in which careful blood-examination fails to disclose the *plasmodium malarix*. The same characteristic marks the temperature-curve in those rare instances of typhoid fever which occur in subjects previously infected with malaria. These two classes of cases run a favorable course as a rule.

Sudden deep temporary drops in the temperature may occur during the fastigium. (1) This may take place during the early part of the fastigium without obvious cause. (2) Intestinal hemorrhage almost invariably produces a sudden, and sometimes a great, fall of temperature. Osler has reported a case in which a drop of 10° F. (5.5° C.) followed melena. The blood does not appear in the evacuations of the patient for six to twelve hours or more after the temperature has begun to fall; and hence a critical decline of temperature during the latter part of the second and the third week of the disease suggests that hemorrhage has probably taken place. (3) The occurrence of *peritonitis* is marked by a sudden and considerable fall of temperature. (4) In the female, abortion or premature delivery occurring in the course of typhoid fever produces a decided lowering of the temperature. (5) Collapse of the circulation sometimes occurs with a notable remission of temperature—an ominous association of events, and one which I observed in two cases occurring in females in the Medico-Chirurgical Hospital. In one of these cases two such periods of collapse occurred, and in the other three, though both finally recovered under prompt and continuous stimulation. Occasionally *hyperpyrexia* is observed in typhoid fever, and most frequently just before dissolution, when the thermometer may register 108° or even 109° F. (42.7° C). A fresh rise with marked irregularity of temperature may occur during the latter part of the fastigium or the period of decline, and is often dependent upon some local complication (late pneumonia, parotitis, etc.).

The stage of defervescence is sometimes much prolonged, though most frequently there is simply a slight evening elevation (99° to 100° F.—37.7° C.), the morning temperature being normal. The causes of retarded decline are, for the most part, obscure. I believe that many of them are ascribable to a mild grade of auto-infection, and in my hands a mild saline laxative has been the means of cutting them short in a number



of instances. An examination should, however, be made for some localized inflammatory complication, though this is not always discernible, as in the case of suppuration in the mesenteric glands, etc. Sluggish typhoid ulcers, which refuse to heal promptly and are due to the now well-known post-typhoid anemia, may act as a cause of the slow decline.

*Post-typhoid Elevations of Temperature.*—After both the evening and morning temperatures have become normal, fresh temporary elevations ( $102^{\circ}$  or  $103^{\circ}$  F.— $38.8^{\circ}$  or  $39.4^{\circ}$  C.) frequently appear. They are, as a rule, unassociated with any other symptoms, and at the end of a few days the temperature falls rapidly to the normal. These are termed *recrudescences*, and are to be distinguished from true typhoid relapses. They are probably produced in various ways—by errors in diet, constipation, mental emotion, excitement, etc.—and there are cases in which the presence of the fever seems to be really a nervous phenomenon (Osler). It is most common in children and in persons of a decidedly nervous temperament. Certain local sequelæ may cause post-typhoid fever, such as abscess, periostitis, etc. Rarely during convalescence a sudden and marked elevation of temperature, accompanied or not by rigor, occurs, but it is usually of short duration and seldom is of serious import. I recently saw, with the attending physician, Dr. Modell, a case in which the temperature had been normal for six days, when rigors, followed by steep elevations of temperature, occurred several times and at intervals of thirty-six or forty-eight hours. These high temperatures were followed by a rapid decline to the normal, and by sweating, leaving the patient profoundly exhausted. Subsequently the convalescence was slow, but uninterrupted.

*Afebrile Typhoid.*—As the term indicates, typhoid fever may run a course attended with all of the characteristic symptoms save the fever. Cases of this kind are of great rarity.

(b) *Skin.*—The *eruption* is highly characteristic, and usually decides the diagnosis. It makes its appearance on or about the eighth day, and sometimes a little later. Occasionally it does not appear until the tenth or twelfth day of the disease. It consists of distinct, rose-colored, and slightly elevated papules, having a rounded or lenticular form and a diameter varying from one or two to three lines. The papules are almost invariably found upon the trunk, and especially upon the upper part of the abdomen and the lower part of the thorax, to which regions they may be wholly confined. They may, however, be absent from the usual seats and present elsewhere, so that the sides of the trunk, the back, and the thighs should always be inspected. They disappear upon pressure, but reappear promptly when pressure is removed. These rose-colored spots last three or four days, and appear in successive crops, each one being made up, usually, of a few spots—a half-dozen to a dozen. Rarely the eruption is abundant on the trunk, even extending to the extremities and head; but there is no direct correspondence between the extent of the eruption and the severity of the cases. Occasionally the spots are entirely absent—a condition most frequently met with in children, and less often in elderly persons.

Other eruptions are often present, and their negative diagnostic value must be kept in remembrance. Minute pearly vesicles (sudamina)



may appear. They are limited to the abdomen, the axilla, and to the inner surface of the thighs as a rule, and are in great measure due to profuse sweating.

A scarlet-colored erythematous eruption sometimes appears at a comparatively early period in typhoid fever, distributed chiefly over the abdomen and chest, and rarely spreading to the extremities as well. Urticaria and petechiæ are rarely seen, though the latter may occasionally result from the transformation of a rose-colored spot. Extensive ecchymoses may occur, but are rare, and merely symptomatic of the hemorrhagic diathesis. Cutaneous boils and abscesses due to secondary infection with the pyogenic coccus are a comparatively frequent and late development in the course of the disease. *Pelionata typhosa* in the form of little bluish subcuticular spots (the "tâches bleuâtres" of the French writers) may appear, but they are not related specially to typhoid fever, and in a recent case of my own were undoubtedly due to pediculi. Gangrene, chiefly of the lower extremities, has been noted in a few instances, and is probably due to an obliterating endarteritis, thrombosis, or embolism.

*Profuse sweats* form a conspicuous symptom in many epidemics of the disease, with or without accompanying fits of chilliness or rigors, and mark the sudoral form of typhoid fever (Jaccoud). Some of these instances closely resemble ordinary intermittents (*vide infra*, Diagnosis). Edema of the skin is sometimes observed and is due most frequently to anemia or cachexia, though sometimes to nephritis. A local form of edema affecting the leg is not uncommon, and for this form thrombosis of the femoral vein is chiefly responsible. A peculiar "musty" odor is exhaled from the skin in some instances of typhoid fever, and mainly in those whose skin-surface has been more or less neglected. In all such cases, and in all cases of prolonged duration, *bed-sores* are likely to develop. They are most prone to occur on the nates and the heels, and, once started, they are apt to spread till they attain to large dimensions, with extensive undermining of the skin. The condition is now serious, and may be the cause of an unfavorable termination, even after the patient has successfully resisted the fever. During and after the conclusion of convalescence the hair falls out, but, fortunately, it is invariably renewed. The nails sometimes become roughened and brittle, and in rare instances drop off, while transverse pale lines or ridges can usually be observed in them, marking the impairment of nutrition during the disease (*vide Relapse*).

(c) **Digestive System.**—The symptoms referable to the gastro-intestinal canal, though not very striking in most cases, are of the utmost importance and interest because of their direct connection with the pathognomonic lesions of typhoid. Beginning with the intestinal canal, and thence proceeding to the symptoms presented by the stomach, spleen, liver, throat, and mouth, will be a natural and convenient order in which to study the symptoms connected with the alimentary tract.

At the onset of typhoid fever *constipation* is the general rule, and this may persist to the end of the illness, though more commonly a moderate diarrhea appears. During the second week of the affection the stools number, on the average, from two to four or more daily. It is only in comparatively rare instances that ten or twelve or more move-

ments per diem occur, the severity of the diarrhea depending more upon the degree of catarrh, particularly of the large intestine, that may be present, than upon the degree and extent of the ulcers. When, however, as rarely happens, the ulcerative process is chiefly limited to the colon, it is an important factor in the production of the diarrhea. Indeed, in those instances—not altogether rare—in which there is urgent diarrhea of a dysenteric character, the ulcers are especially marked in the colon, with diphtheritic inflammation of the mucosa as a frequently associated lesion. Involuntary discharge of the feces may occur.

The *stools* present a characteristic yellow appearance, suggesting by their color and consistence a comparison with pea soup. They are usually either fluid or of the consistence of jelly, and are offensive and of an alkaline reaction. On standing they separate into two layers—an upper, liquid, cloudy layer, and a lower, thick yellow, sedimentary layer, in which, on macroscopic examination, remnants of food and grayish yellow fragments (necrotic crusts of Peyer's plaques) from a half to an inch in length may be detected. Microscopically, they have been found to contain undigested particles of food, epithelial debris, blood-corpuscles, crystals of triple phosphates in abundance, and innumerable bacteria. Laboratory experimentalists have been able to demonstrate the presence of the typhoid bacillus in the dejecta. *Tympanites*, mainly affecting the colon, is a common though rarely a striking feature, and cases of the most serious nature are observed in which the abdomen presents a concavity throughout the entire illness. The latter is less unfavorable, by far, as a symptom than excessive tympanites, which interferes with both the respiration and heart action. Tympanites is apt to be most marked in serious cases which have diarrhea as a prominent symptom, though the latter may not even be present. It is due to the generation of gas from decomposing food, and to the arrest of peristaltic movements in consequence of the degeneration of the muscularis of the intestines. Pain is absent in the majority of cases, and when present is not intense, save in rare instances. Pressure upon the ileocecal region usually causes a gurgling noise, but, although this symptom is commonly present, it is not characteristic of the disease. There is generally also a slight degree of tenderness of the abdomen under pressure, most marked in the right iliac fossa, and hence, in all probability, due to the presence of ulcers in this region. Absence of tenderness, however, is not a safe indication of the absence of extensive ulceration. Extreme sensitiveness generally denotes peritonitis (often without perforation), though the symptom may be quite well marked as a result of constipation only.

*Intestinal hemorrhage* occurs in from 4 to 7 per cent. of cases, its frequency varying with different epidemics. The hemorrhages appear almost invariably during the latter part of the second and third week, being caused by the opening of blood-vessels during the necrotic or ulcerative process. Bleedings may also take place from the soft and hyperemic edges of the ulcer (*vide supra*), and when it occurs quite early in the disease it may be in consequence of an excessive hyperemia of the lymph-follicles. The amount may be so small as to be scarcely discernible by the naked eye, or it may be from one to two or three pints (0.5–1.5 liters), or even more. In one of my own



cases at the Medico-Chirurgical Hospital the total amount of blood discharged from the bowel was  $78\frac{1}{2}$  ounces, or very nearly 5 pints (2.5 liters), and yet the patient recovered. The blood presents a dark hue, and that which is passed last may be tarry.

The significance of intestinal hemorrhage, however slight, is always grave. On the other hand, recovery is possible even if the hemorrhage be copious and oft-repeated, as is shown by the case before cited from my own experience; and in general terms it may be said that death supervenes in from 30 to 40 per cent. of all cases. A fatal result may occur as the direct effect of a sudden profuse hemorrhage. When death does not follow immediately, however, the signs of collapse (more or less intense) and of anemia appear; yet intestinal hemorrhage sometimes exerts a favorable influence, and particularly on the temperature and nervous symptoms. In a couple of instances I have observed stupor and delirium quickly giving place to perfect consciousness. Lastly, when typhoid fever occurs in the hemorrhagic diathesis melena may manifest itself in connection with hemorrhage from other outlets of the body.

*Perforation*, which almost invariably produces fatal diffuse peritonitis, is the accident most to be dreaded. It does not bear a fixed relation to the severity of the affection, but in the 2000 Munich cases (*vide supra*) perforation occurred in 114; and according to Fitz, who tabulated 4680 cases of typhoid fever, there is a mortality of 6.58 per cent. from perforation of the bowel. It is much more common in males than in females, and appears in a ratio of about 71 to 29. Age has a decided influence, the complication being most marked between ten and forty years old, whilst in children it is rare; and, though perforation may occur at any time in the course of typhoid fever, it is most common between the second and fourth weeks of the disease. In the cases analyzed by Fitz perforation was found in the ileum in 81.4 per cent., in the large intestine in 12.9 per cent., in the vermiform appendix in 2.5 per cent., and in the jejunum in 1.29 per cent. The accident is usually announced by the sudden advent of acute pain in the abdomen, quickly followed by the symptoms of collapse; and the fact that diffuse peritonitis, following perforation, may develop insidiously must be recollected. The abdomen becomes greatly distended as a rule, and is exquisitely tender to the touch. Fluctuation can sometimes be elicited. On *percussion* splenic and hepatic dulness are often absent, and in this connection there is danger in making an error in diagnosis, since hepatic dulness is also wanting when the distended intestines lie in front of the liver. The general collapse of the circulatory system is evidenced by the pinched features, hollow cheeks, vomiting, and the small, frequent pulse.

No other complication is so grave as peritonitis. Its causes have been pointed out previously (*vide p. 21*), but from a clinical point of view a division of all the cases into two classes—those due to perforation and those due to other agencies—is desirable. The instances that develop independently of actual perforation are not of infrequent occurrence. They usually assume the local or circumscribed form of peritonitis, which is occasioned by direct extension of the inflammatory process from the intestinal ulcers. The condition presents corresponding



areas of tenderness under gentle, and especially under prolonged, pressure. It is, however, confessedly difficult to diagnose between the intra- and extra-intestinal states, which are accompanied by sensitiveness to gentle palpation, particularly when peritoneal inflammation exists in a mild form. Generalized peritonitis may succeed to the circumscribed variety in consequence of the extension of inflammation to the peritoneal sac, and without perforation.

The *mesenteric lymph-glands* may soften or suppurate (*vide* Pathology), and, as before mentioned, may be the exciting cause of a recrudescence, or they may rupture and cause diffuse peritonitis.

*The Spleen.*—With few exceptions the spleen is enlarged in typhoid fever, the edge usually being palpable below the margin of the ribs, on or before the commencement of the fastigium. It generally goes on increasing in size till near the beginning of the third week, and lessens during the latter part of the third and fourth weeks. In four of Osler's autopsies it weighed less than normally. Swelling of the spleen is sometimes absent after a copious intestinal hemorrhage, as well as in elderly typhoid subjects. As before mentioned, the enlargement in many cases is not demonstrable by percussion when the tympanites is excessive, but by means of careful palpation we can in most cases satisfy ourselves of its existence or non-existence, despite the great distention of the bowel. Suppurative infarcts, or softening of the spleen may start a peritonitis. Rarely, rupture of the organ may occur, which is manifested usually by intense pain in the splenic region. A slight swelling of the *liver* can sometimes be detected, and as a rule parenchymatous degeneration takes place. Among the least frequent of complications is jaundice, and abscess of the liver also rarely occurs.

*The Stomach.*—The stomach offers no characteristic symptoms. Of the anorexia, which is constant until recovery begins, enough has been said, but during convalescence the appetite returns, becoming even voracious. Nausea and vomiting may occur during any stage of the disease, and are most common at the beginning, but when they appear as late symptoms they are probably excited either by gastric ulceration or by peritonitis. Nausea is usually traceable to definite causes—either to errors in diet or to the use of irritating medicaments, but that vomiting does occur from unknown and inevitable causes in very rare instances I am fully convinced. This sort of vomiting was present in the afore-mentioned case reported by myself in which there were profuse hemorrhages. It may become a serious or even fatal symptom.

*The Pharynx.*—The pharynx frequently shows catarrhal irritation, and the patient may complain of dryness or a burning sensation in the throat. Actual sore throat may be present at the time of onset, and this may be associated with a diffuse erythematous rash, suggesting scarlatina.

*The Tonsils.*—There is a special form of typhoid—*tonsillo-typhoid* or *pharyngo-typhoid*—in which there appear upon the tonsils peculiar patchy elevations, whitish in color, which undergo subsequent ulceration. It is not improbable that these lesions result from the local action of the specific bacillus in an unusual situation. *Thrush*, affecting the mouth, throat, and even extending to the esophagus, not infrequently arises as a complication. The tongue is heavily coated, as a rule, with

a yellowish-white fur; later it clears off near the edges and tip, while the center becomes dry or brown and sometimes fissured. The lips are also dry, sometimes fissured, and often covered with dry, black crusts (*sordes*). *Ulcerative stomatitis* may occur if the mouth be not kept clean. Under these circumstances secondary lesions evincing unpleasant and even serious symptoms may also arise in organs more or less remote from the mouth, and among these is *parotitis*, which is most probably caused by the staphylococcus or streptococcus reaching the parotid gland by way of Steno's duct. The condition is betrayed by such symptoms as pain, redness, and finally by fluctuation, with an elevation of the bodily temperature. It is a late-appearing development, and is usually unilateral, though it may be bilateral. *Suppurative otitis media*, a rarer complication, arises in a similar manner, the pathogenetic agents passing from the throat to the ear through the Eustachian tube.

(d) **Respiratory System.**—As pointed out in the section on Pathology, bronchitis is almost invariably present, but in the majority of instances the cough is slight. The condition is recognized by the existence of numerous sibilant râles. Very rarely is it a striking feature in the early stage of typhoid fever, and then, except this fact be remembered, room for error of diagnosis exists. Moreover, in cases that are improperly treated the bronchial secretions are apt to accumulate, and a well-marked bronchitis may be the result. It may be said, however, that, as a rule, bronchitis does not assume a severe type in cases which receive proper attention from the beginning, provided the patient be not unusually stupid or unconscious. When the nervous phenomena are pronounced, however, and the patient maintains the dorsal decubitus (expectorating little or nothing), bronchitis of a severe grade and affecting the smaller bronchi is almost inevitable. The occurrence of an intense generalized bronchitis is also favored by certain other conditions, such as corpulence, advanced age, emphysema, etc.; and these are the cases that are apt to lead to lobular infiltration—the so-called aspiration pneumonia.

*Lobular pneumonia* may take on a putrid nature and the consolidated area may become gangrenous. As a sequel, pleurisy with effusion or empyema may originate in consequence of the infiltrated lobules being contiguous to the pleura. If these lobules, occupying the periphery of the lung, become gangrenous, perforation of the pleura, leading to pyopneumothorax, may result. Lobular pneumonia may be attended with hurried breathing or troublesome cough. More commonly, the local symptoms are either altogether wanting or feebly marked, and this is especially true of the severer forms of lobular pneumonia, which occur in patients in whom profound nervous prostration coexists with more or less complete unconsciousness. Sole reliance is to be placed upon the results of a physical examination, which even in the absence of subjective symptoms should be repeated daily. Points or surfaces of dulness, most marked near the bases of the lungs and frequently on both sides, are found on percussion. Fine moist râles, most marked toward the bottom of the thorax, form a very characteristic sign, and are heard in every direction on auscultation. In order to ensure a certain diagnosis of lobular pneumonia both the circum-



scribed dulness and moist râles must be found present in the same situation.

*Lobar pneumonia* is a not uncommon complication. In a small percentage of cases it develops early, and is most probably the result of a special concentration of the poison in the lungs, giving rise to the so-called pneumo-typhoid fever (*vide infra*, Varieties). These cases are often mistaken for primary lobar pneumonia. Their onset may or may not be marked by a rigor, but it is usually more gradual than that of primary lobar pneumonia. Characteristic typhoid symptoms soon follow, and at the end of the first week or thereabouts the pulmonary symptoms gradually abate, while those most characteristic of typhoid (enlarged spleen, roseate spots, etc.) occupy the foreground. Lobar pneumonia more often develops as a late complication—in the second or third week, or even during convalescence—but it is not attended by the usual phenomena (rigor, cough, rusty expectoration, intense chest-pain, etc.), and hence may be easily overlooked. The temperature may be either quite elevated or at times only moderate. The diagnosis is to be made from the physical signs, together with the peculiar temperature-curve, which may present marked irregularities. Pulmonary infarction and abscess of the lungs are occasional complications.

*Hypostatic congestion of the lungs*, due to enfeeblement of the cardio-pulmonary circulation, is a comparatively frequent concomitant, appearing in the third week of the disease. It is generally bilateral, affecting the base of the lungs, and is promoted by the effects of gravitation. It is almost always associated with more or less edema of the lungs. The subjective symptoms, including fever, are usually negative, while the objective signs are those of partial or complete consolidation of the bases (defective resonance or dulness, broncho-vesicular breathing, with moist râles). *Miliary tuberculosis* rarely develops as either a complicating affection or, it may be, as a sequel. A spasmodic or jerking inspiration when pneumonia does not exist is a precursor of coma (Flint).

*Laryngitis*, indicated by hoarseness, is an occasional complication. The laryngeal ulcers may extend in depth to the perichondrium, and in this way may rarely promote that grave though not necessarily fatal condition, *perichondritis* with edema of the glottis. The symptoms of laryngeal stenosis are apt to develop.

*Epistaxis* appears early in a large number of cases, and is a valuable diagnostic symptom. It may also occur during the fastigium, and particularly toward the latter part, when it is of little or no diagnostic, but of grave prognostic, significance. It is apt now to be troublesome, and may even, as in a case I saw recently with Dr. I. Newton Snively, be so persistent as to lead to a fatal issue.

(e) **The circulatory system** presents no characteristic symptoms. The heart-sounds are but little affected, as a rule. In cases of asthenic type and in severe typical instances the first sound of the heart may grow quite feeble and ultimately resemble the second (embryocardia). Under these circumstances a soft systolic murmur may be faintly heard along the left border of the sternum. Among occasional complications presented by the heart is pericarditis, and still less frequent is endocarditis. Myocarditis is somewhat more common. The sudden development of circulatory collapse in the course of typhoid fever, as previously noted,



may be due chiefly to myocardial inflammation; and there may be a brief though alarming derangement of the heart action, due to functional disturbances of the sympathetic and pneumogastric nerves.

The *pulse* is accelerated, but not, as a general rule, in proportion to the height of the temperature until late in the affection. Its average rate is from 84 to 108, but it may go much higher, and when the pulse is maintained at 130 or more for days together it is of ominous import. The temperature, moreover, may be of average height, while the pulse is normal or only slightly quickened throughout; and hence the increase in the pulse-rate cannot be due solely to the elevation of temperature. As before intimated, the extreme debility which comes on during the third week in severe cases may have, as one of its manifestations, a very rapid pulse, reaching to 160 or more (the so-called running pulse), and with or without marked irregularity. Slight irregularity is sometimes observed, either during the height or decline of the affection, but as a rule this soon disappears, and proves of no serious consequence. Marked temporary accelerations are often caused by undue exertion or mental excitement. The lowered arterial tension is shown by a dirotism of the pulse—a symptom which is not characteristic of typhoid fever, however, since it is well marked in other acute infectious diseases, though less frequently. During convalescence the pulse often becomes sub-normal in rate, and bradycardia is oftener a sequel of typhoid than of any other acute infectious disease.

*Venous thrombosis* occurs in 1 per cent. of all cases (Murchison). Its most frequent seat is the left femoral, and the next most frequent the right femoral vein, and it is the immediate result of cardiac weakness, except perhaps in those rare instances that arise early in typhoid. For the latter no definite cause has as yet been found. Coming on, as it usually does, during convalescence, it manifests itself by swelling and edema of the extremity affected. There are pain in the thighs and calves, and tenderness (on pressure) over the course of the femoral vein, and often in the region of the calf of the leg as well. It causes fever of a moderate grade and irregular type, and then in the course of from two to three weeks the swollen member may be reduced to its normal dimensions. This complication is usually not of a serious nature. Occasionally, however, clotting extends into the pelvic veins, or even into the vena cava, when the condition becomes more serious, and sudden death has resulted from the detachment of emboli. The thrombus may undergo suppuration, to which systemic septic infection may be a secondary event.

*Thrombosis*, and less frequently embolism, in the arteries, combined with renal, splenic, and pulmonary infarcts, may be encountered in typhoid fever.

The large or small arteries may become obliterated, either by embolism or thrombosis, in extremely rare instances, but whether the thrombosis under these circumstances is brought about by a peculiar condition of the blood which favors clotting, or by a localized arteritis, or in consequence of the operation of these combined factors, is not definitely known. If, as is usual, the femoral artery be involved, the blood-supply to the foot and leg is cut off and gangrene of those parts must follow. The condition may be bilateral. It may be detected

early, owing to the absence of a femoral pulse, before the signs of gangrene appear, but the condition is highly dangerous. Recovery ensues in perhaps more than half of the cases.

The *blood* presents certain changes, some of which are valuable for diagnostic purposes. In those rare cases in which copious diarrhea or profuse sweats are present the red corpuscles may be relatively increased in number during the febrile period, owing to loss of water. There is, however, in the majority of instances, little or no decrease in the number of red corpuscles till the end of the second week. They are markedly diminished, as a rule, during convalescence. Indeed, the oligocythemia may attain to an immoderate degree. In one of Osler's cases the number of red corpuscles was as low as 1,300,000 per c.mm., but I have personally never found the blood-count under 1,800,000.

There is a greater relative decrease in the amount of hemoglobin than in the number of red corpuscles, and the restoration of the hemoglobin in the convalescent period takes place more slowly than that of the red corpuscles. The number of white corpuscles remains at or a little below the health standard until late convalescence, when it sinks to a moderate degree—furnishing a count of about 2000 per c.mm. This fact is an important aid in the differentiation of typhoid fever from

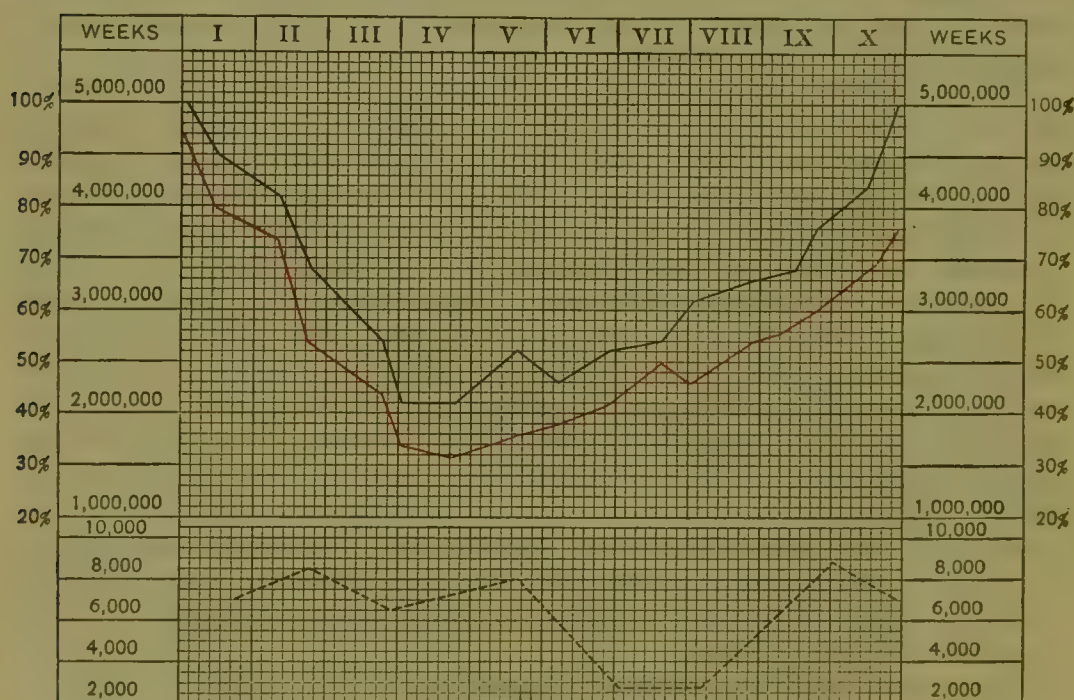


FIG. 4.—Chart illustrating the blood-changes in typhoid fever: upper curve, red corpuscles; middle curve, hemoglobin; lower curve, white corpuscles.

acute inflammations and infectious (febrile) affections accompanied by exudation, in which leukocytosis is marked, and from all suppurative processes in which the polynuclear neutrophils are moderately increased. In typhoid fever there is also a relative preponderance of the mononuclear forms in addition to the absolute decrease of the leukocytes. The blood-characters in typhoid are shown in the accompanying chart (Fig. 4).



(f) **Nervous System.**—The persistent headache that is almost always present is among the most prominent symptoms during the first week, but it diminishes steadily during the early part of the second, as a rule. It affects the temporal, occipital, and cervical regions, and when the onset is comparatively sudden, pain in the back is also a more or less conspicuous feature during the first few days of the illness. In a small class of cases, however, the effects of the typhoid bacilli or their toxins are manifested solely in the nervous system from the very onset. In such there are violent headaches, retraction of the head, rigidity, photophobia, and muscular twitchings (rarely convulsions)—all of which symptoms indicate meningitis. The diagnosis of meningitis as a complication must be made with extreme caution, since, no matter how complete the clinical picture may be, the post-mortem examination usually reveals a total absence of meningeal inflammation. It must not be forgotten, however, that meningitis is one of the rarest of the complications of typhoid fever. *Vertigo* may accompany the headache, but it seldom outlasts the latter. Before delirium manifests itself wakefulness and restlessness at night are very annoying, and later the same symptoms may be observed associated with the delirium. In cases of moderate severity mental dulness, and even actual hebetude, are almost invariably present. Questions are apt to be answered inconsistently and in monosyllables, and the patient sleeps poorly, notwithstanding the pseudo-somnolent state in which he almost constantly appears.

*Delirium* is frequent in the severer cases. It is, however, not an uncommon event for those of moderate severity to be free from this symptom throughout the attack. It is, as a rule, most marked at night or at some time when the patient is left alone. His delusions may impel him to attempt to leave his bed, but more commonly there is mild or noisy delirium, with more or less restlessness. He may lie somnolent, soliloquizing in a loud whisper (muttering delirium), and this so-called *typhomania* may gradually give place to actual coma toward the close of the middle period of the disease. In not a few cases—mild or severe—coma is developed suddenly, and is often a mortal symptom. Still another unfavorable sign is a picking at the bed-clothes or a grasping at imaginary objects (*carphologia*).

The delirium may assume an hysteric type, the patient usually exhibiting the saddest emotions, and if he be an alcoholic he may be seized with delirium tremens. In a case of typhoid fever that I saw recently with Dr. S. W. Morton hysteric delirium developed during convalescence, but did not last more than twenty-four or thirty-six hours.

The *motor nerves* also present notable disturbances in association with the sopor and the forms of delirium previously described. Slight twitchings of the muscles of the face and extremities are quite common, and when they affect the tendons of the wrist and fingers the term *sub-sultus tendinum* is applied. The lips, tongue (especially when protruded), lower jaw, and even the extremities, are often in a state of constant tremor. During this motor irritability the reflexes are increased, but when profound coma comes on they are either largely diminished or totally abolished. The toxins and chemical secretions of the typhoid bacillus, acting poisonously upon the nervous centers, are undoubtedly the cause of the nervous symptoms in typhoid.



*Nervous complications and sequelæ* may arise. Chief among these is paralysis, which is most probably due to neuritis. The lesion may involve one, two, or more nerves, and in this way we may have either a paralysis of one limb or, more rarely, a true paraplegia. *Aphasia* may be a sequel, particularly in children. *Hemiplegia*, due to hemorrhage or a localized encephalitis, may occur either as a complication or sequence of the disease. Following typhoid fever, the patient may exhibit evidences of *mental enfeeblement*, and even *insanity* where a predisposition to this condition has existed; and insanity is relatively more common after this disease than after any others belonging to the same class. I have seen two instances, both of which recovered, while Osler has seen five, four of which ended similarly. It is in most cases, as pointed out by Wood, a confusional insanity, due to exhaustion and impairment of the nutrition of the nerve-centers, while in a smaller contingent it takes the form of a true melancholia. After the conclusion of typhoid, as well as during its course, *neuralgia* affecting the occipital and other cranial nerves is not infrequent. Great hyperesthesia of the skin and muscles is common during convalescence, attacking the lower extremities by preference (Strümpell). The so-called "typhoid spine" (Gibney) has also been observed, and consists in an acute inflammation of one or more vertebræ following typhoid. The chief symptoms are pain in the back and hips of a lancinating character. The point of origin appears to be the small of the back; thence the pains extend paroxysmally up and along the spine and to the abdomen. They subside gradually, leaving the back weak and painful on attempts at turning in bed, etc. Plantar and other skin-reflexes increase, and the knee-jerks are preserved.

(g) **The Urinary System.**—*Urine.*—The urine is lessened in quantity and high-colored, with an increased specific gravity up to the arrival of the stage of decline. About this time, and rarely earlier, it grows light in color, larger in quantity than the normal, and the specific gravity is relatively diminished. Both urea and uric acid are increased during the earlier stages, and sometimes throughout the attack, while during convalescence both are diminished. On the other hand, the chlorids are diminished during the active stages of the disease and increased during its decline. Afebrile albuminuria is quite common, but is of no clinical importance.

*Acute nephritis* may develop as a complication in the earlier or later course of the disease, and can be recognized to a certainty only by a thorough appreciation of the urinary phenomena. The urine is diminished in quantity, being often scanty, and there may be retention. It contains characteristic morphologic elements (albumin, casts, blood, and epithelium). The development of the typhoid state in this affection is rendered much more probable in the presence of this complication, and, moreover, uremic symptoms often put in an appearance at this juncture, and then the situation is really serious. Acute nephritis may arise at one or other of three different periods, and its significance varies with the time of onset: (a) at the beginning of the fever, when it often obscures the true nature of the malady. This is the *nephro-typhoid* of the German authors, and will be referred to hereafter (*vide infra*, Varieties); (b) in the early part of the fastigium or the second week of the

disease. Coming on at this time—an event which I have observed in two instances—its relation to the typhoid bacillus or its toxin is not definable. It is probable, however, that it is to be ascribed to the local effect of the toxin upon the renal tissues. Both of my own instances proved fatal, and in both an autopsy was refused. Wagner<sup>1</sup> has had 5 cases of recovery in succession, but the high mortality mentioned by Amat—10 deaths in 12 cases—is the more common experience. (c) Acute nephritis may arise as a sequel of typhoid, when, with the usual symptoms of acute nephritis, there is almost invariably associated a decided edema. In this category of cases recovery is to be expected. The lymphomatous nephritis of Wagner (*vide supra*, Pathology) is usually without symptoms.

*Diabetes mellitus* is, in extremely rare instances, developed after typhoid. Hematuria has also been observed as an occasional symptom of the hemorrhagic diathesis.

The *diazo-reaction* of Ehrlich is an aid in diagnosis, but, unfortunately, may be present also in acute phthisis, meningitis, measles, and other acute infections attended with fever. To obtain it two solutions (*a* and *b*) are needed: We mix 1 part of solution (*a*), which consists of a 0.5% solution of sodium nitrite, with 40 parts of solution (*b*), which consists of 2 grams of sulfanilic acid, 150 c.c. of hydrochloric acid, and 1000 c.c. of distilled water. To this an equal volume of urine is added, and the contents of the test-tube are then thoroughly shaken. A layer of ammonium hydrate is now superimposed, and at the line of contact a ruby or pink ring develops. I have found the reaction rarely absent during the fastigium or after the eighth or ninth day. A brownish ring is given by normal urine.

There is a post-typhoid, diphtheritic *pyelitis* in which the pelvis and calices of the kidneys are primarily the seat of membranous exudation, and later of erosion and ulceration. The urine generally contains blood and pus. Osler has met with this condition in 3 autopsies, in 1 of which it was associated with extensive membranous inflammation of the bladder.

Simple *vesical catarrh* is a rare complication except as the result of catheterization for retention.

*Orchitis* and *ovaritis* are occasional sequels.

(h) **The Joints.**—*Monarticular arthritis* may arise as a complication, and often proceeds to suppuration. *Polyarthritis* also occurs, but it is by no means so common. These conditions are due to accidental infection.

(i) **The Bones.**—*Periostitis*, leading to *necrosis*, is a not very rare sequel of typhoid. The favorite seat is the tibia, though in a case of typhoid under my own care at the Philadelphia Hospital it affected the os calcis. *Osteomyelitis* may also occur.

(j) **The Muscles.**—As in the case of the heart, so the voluntary muscles exhibit hyaline degeneration; and abscesses, in consequence of secondary infection or of infection with the typhoid bacillus itself, may be located in the muscles.

**Associated Acute Infectious Diseases.**—Malarial fever may be combined with typhoid, though the relationship is not a vital one. In an

<sup>1</sup> *Deutsche Archiv für klin. Med.*, Bds. xxv. and xxxvii.



analysis of 2122 cases of malaria typhoid fever was associated in 8.<sup>1</sup> Many instances of so-called typho-malarial fever would be shown to be pure typhoid by a careful blood-examination, as the presence of chills, sweats, and an intermittent temperature-curve are sometimes observed in this disease (*vide supra*).

**Pseudo-membranous inflammation**, as above intimated, may occur in the naso-pharynx, larynx, gall-bladder, and genitals. *Measles*, *scarlatina*, and *chicken-pox* have also been known to arise in the course of, or during convalescence from, typhoid fever.

**Erysipelas** is a rare secondary affection coming on either during the height of the affection or (more frequently) after its close. Typhus fever may be associated with typhoid, but is an exceedingly rare occurrence.

**Clinical Varieties of Typhoid Fever.**—These are numerous, and may grow out of peculiarities manifested during the course of the affection, as may be observed not only in different epidemics, but also in the same epidemic. The groups of cases described here have reference particularly to the degree of severity of the type, which varies between the wide limits of extreme mildness on the one hand and extreme severity on the other. The course of the disease may also be modified by the occurrence of one or more of its manifold complications.

(1) **The Mild or Rudimentary Form (Typhus Lævissimus).**—Of this variety many cases occur, and especially among children. The characteristic typhoid symptoms are scanty, and at times even entirely wanting. The spleen is almost always enlarged, the roseate spots are sometimes present, while the temperature is moderately elevated and often partakes of the same character as that of true typhoid. The fever, however, may pursue the remittent type. Complications presented by special organs are usually absent, but grave accidents (intestinal hemorrhage, perforation) are not impossible.

The diagnosis is always difficult, owing to the feeble development of the characteristic symptoms, and in the total absence of the latter is out of the question; but the recognition is greatly aided if a causal connection between them and typical cases can be shown to exist with great probability.

(2) **The abortive form** has a sudden onset, and is often marked by fits of shivering. The characteristic features of the disease (enlargement of the spleen, abdominal symptoms, rose spots, etc.) appear earlier than in the usual type, and soon become quite well marked. The fastigium is short, and the temperature, from the seventh to the twelfth day of the illness, declines by a prompt lysis, with profuse sweating. With the rather rapid fall of temperature there is a no less rapid improvement in every other leading symptom. Convalescence is speedy.

(3) **The Ambulatory Form (Latent or Walking Typhoid).**—The patient continues to walk about, either experiencing but slight disturbance or being unwilling to take to his bed. Such cases do not come under the care of the physician in many instances. Others, on account of debility, anorexia, diarrhea, and other vague symptoms, finally consult their physician, who may discover the presence of all the characteristic

<sup>1</sup> "The Complications of Malaria," *Journal of the American Medical Association*, vol. xxiv. p. 919, by the author.



features of the disease. A third contingent, belonging to this form, continue to move about, or even to follow their usual vocations, till seized suddenly with profuse intestinal hemorrhage or general diffuse peritonitis following perforation. The likelihood of these grave developments is much greater in the case of persons who go about or travel long distances while suffering from this disease.

(4) **The afebrile** is an exceedingly rare form of the affection—in this country at least. Liebermeister, however, has met with a number of cases at Bâsle, the symptoms being lassitude, depression, headache, neuro-muscular pains, anorexia, slow pulse, furred tongue, constipation or diarrhea, with enlargement of the spleen and roseate spots. These cases are often confined to bed, and there are occasional attempts at evening exacerbations of temperature ( $100.5^{\circ}$  F.— $38^{\circ}$  C.). Sub-normal temperatures are sometimes associated, but I have seen only a single instance that I regarded as belonging to this form.

(5) **Severe or Grave Forms.**—These may be dependent either wholly or in great part upon the degree of virulence of the typhoid poison. Under these circumstances there will be a profound intoxication of the system, as shown by high temperature, violent nervous symptoms, and great prostration. The grave types may arise in the course of cases of average severity from the development of serious complications. Again, to serious forms belong those cases that begin with the characteristic symptoms of a localized inflammation—*e. g.* the *cerebro-spinal form*, in which the nervous symptoms greatly predominate at the onset; the *nephro-typhoid* (before alluded to), in which the preliminary symptoms are those of acute Bright's disease; the *pneumo-typhoid* (*vide supra*), which begins with the manifestations of a more or less frank pneumonia.

*Pleuro-typhoid.*—The cases begin as an acute pleurisy, having its special characteristics, and these are followed, soon or late, by the diagnostic evidences of typhoid fever. Talamon<sup>1</sup> distinguishes these cases from simple pleurisy by the intensity and continuous course of the fever, by the general depression, headache, and vertigo, and by the sleeplessness. Should any doubt remain, it will be dissipated by the eighth or ninth day by the presence or absence of rose-colored spots, enlargement of the spleen, and other features characteristic of typhoid.

The *sudoral form* and *tonsillo-typhoid* (before described) also belong to this category. Many circumstances connected with the individual influence decidedly the general course of the affection, and these may be expressed in part in the several forms following, which are based upon such factors as *age*, *habits*, etc.

(6) **Typhoid Fever in Children.**—The onset is rather more abrupt than in the adult, and certain prodromal symptoms are very generally absent (epistaxis, chilliness, etc.). On the other hand, bronchial and nervous symptoms are often quite pronounced. Again, during the fastigium some of the characteristic features may be missing—*e. g.* diarrhea and tympanites—while the eruption may either be entirely wanting or less copious than in older subjects. Intestinal hemorrhage is rare and perforation almost never occurs, but, as previously pointed out, aphasia is a more common sequel than in adults. The same is true of arthritis and the bone-lesions. The mortality is not over 1 per cent.

<sup>1</sup> *La Médecine moderne*, Paris, 1891.

(7) **Typhoid Fever in the Aged.**—The course of the affection presents no regular type. The temperature is not as high as usual, but there is marked adynamia and serious danger from certain complications, such as pneumonia, nephritis, coma, etc.

The diagnosis is difficult, owing to the prominence of the nervous and pulmonary symptoms on the one hand, and the frequent absence of the more characteristic symptoms of typhoid on the other (rash, enlargement of the spleen, and peculiar temperature-curve).

**Diagnosis.**—Unless all the chief characteristic features be present with a clear history, it is a golden rule not to make a positive diagnosis. Obviously, then, the physician at the first visit cannot, in many cases, diagnose typhoid with absolute certainty. In the majority of instances he is called after the case has progressed to or near the close of the first week. If the case have been a typical one, the history of the gradual development of the disease, marked by such symptoms as languor, anorexia, headache, dulness, slight chills, increasing fever, and sometimes nose-bleed, will be obtained, and justify a strong suspicion of typhoid. When, in addition, diarrhea and the objective symptoms, splenic enlargement, tympanites, gurgling, with tenderness in the ileo-cecal region, are present, the diagnosis of typhoid is made highly probable. After the lapse of a few days—the beginning of the second week—the roseate spots usually appear, and then all doubt is removed. In atypical cases a positive opinion, particularly if the rash be absent, must often be withheld till an advanced stage is reached. In such instances the sudden occurrence of a significant symptom, such as intestinal hemorrhage or a characteristic decline by lysis, is exceedingly helpful. The etiologic circumstances need to be considered carefully, since to show a causal relation between an obscure case and one that is clearly typhoid leaves little to be desired.

Briefly, the most trustworthy diagnostic features are the gradual onset, peculiar temperature-curve (made up of the “step-ladder” stage of development, the continued type of the fastigium, and the decline by lysis), enlarged spleen, and the rose-colored spots.

Investigations by Pfeiffer upon the specific bactericidal substances developed in the blood of animals immunized by injection of typhoid bacilli have furnished a practical and reliable means of diagnosis of this disease from blood-serum. It remained, however, for Widal and others to show that if to a drop of blood-serum, or to a drop of water containing a solution of dried blood from a typhoid patient, a moderate number of typhoid bacilli were added, a very peculiar reaction occurred.

Johnston of Montreal has simplified the technic: The blood is obtained upon a clean glass slide from a needle-prick of the ear or finger of the suspected case. It is allowed to dry, and is then carried to the laboratory. A loop of bouillon-culture of genuine typhoid bacilli is placed upon a clean cover-glass, and to this is added a large loopful of a watery solution of the dried blood-specimen. The cover-glass is inverted over the concavity of a hollow slide and sealed at the edges with melted vaselin. Under the microscope, with a high-power dry lens or with a one-twelfth oil-immersion lens, a rapid clumping of the bacilli in the hanging drop can be observed,<sup>1</sup> and their motions cease almost instantly.

<sup>1</sup> *Medical News*, Nov. 14, 1896.



If this specific reaction is not obtainable in a case sick over a week, typhoid fever may be excluded. It is to be remembered, however, that in persons who have had typhoid fever within ten years the reaction may take place.

The cases that, in the beginning, manifest the special evidences of an infectious disease, with the well-defined local inflammatory lesions previously referred to (tonsillo-typhoid, pneumo-typhoid, pleuro-typhoid, nephro-typhoid, etc.) cannot be recognized at the outset. The same local inflammatory condition may, independently of typhoid fever, be combined with a genuine typhoid state. In all instances of typhoid fever in which, at the time of onset, early localization occurs in the throat, lungs, or kidneys, the general features (degree of fever and prostration) are apt to be out of proportion to the local, and the former are apt to continue to develop after the subsidence of the latter. A careful observation of the symptoms after the first week will detect, either immediately or in the course of a few days, undoubted symptoms of typhoid; and in any acute affection in which the symptoms of the typhoid state coexist with a local inflammatory process the existence of typhoid fever should be suspected and the appearance of the eruption daily anticipated.

Cultures may be obtained by puncturing the spleen, but the wisdom of this procedure is very questionable, since the bacilli may be obtained in quantities from the stools.

**Differential Diagnosis.**—(1) **Typhus fever** is to be differentiated by its appearance as an epidemic, by its sudden onset, by the deeper stupor, the besotted expression of the features, the injected conjunctivæ, the contracted pupils, the appearance on the fourth day of maculæ which are speedily transformed into petechiæ; by the shorter course and the abrupt termination by crisis.

(2) **Acute miliary tuberculosis** has been, and still is, frequently mistaken for typhoid fever. The former is to be differentiated from the latter by the greater frequency of the pulse and respirations, the prominence of the cough, and in some instances by the bloody expectoration; by the pronounced cyanosis, the presence (sometimes) of choroidal tubercles, and the existence (constantly) of leukocytosis, which does not occur in typhoid. *Blood-examinations* have occasionally shown the presence of the tubercle bacillus. There is an absence of the peculiar temperature-curve and also of the characteristic lenticular spots and abdominal symptoms of typhoid.

(3) **Malarial fevers** may assume, more or less nearly, the continued form, and there are also typhoids which affect a remittent or an intermittent type of malarial fever. The latter can always be differentiated by the therapeutic test with quinin, and by a careful blood-examination for Laveran's hematozoa, which are present in the blood.

Should *typho-malarial fever* be suspected, and should the typhoid symptoms be unequivocal, the finding of the malarial organism will differentiate the hybrid from pure typhoid.

(4) **Relapsing fever** is distinguished by its abrupt onset with rigor, high fever, pain in the epigastrium; by the brief duration, termination by crisis, and the occurrence of a relapse at the end of a week; by the absence of the characteristic eruption; by the temperature curve; and

by the marked nervous symptoms of typhoid. The finding of the spirilla, however, reliably discriminates relapsing fever.

(5) **Meningitis.**—In striking contrast with the specific typhoid symptoms meningitis exhibits marked hyperesthesia, intolerance of light and sound, exaggerated reflexes, and often muscular rigidity before the stage of effusion; also restlessness, peevishness (unlike the dulness observed in typhoid patients), vomiting, and constipation (*vide* Acute Miliary Tuberculosis). The temperature maintains a lower level on the average, and is more irregular in type than in typhoid; the pulse is more irregular, and the nervous symptoms assume greater prominence in the earlier stages, particularly headache and delirium. On the other hand, the absence of true typhoid symptoms aids in the discrimination of acute meningitis.

(6) **Tuberculous meningitis** gives a characteristic previous or family history, occurs usually in young subjects, and the tendon and cutaneous reflexes exhibit the widest possible variations as to intensity, within brief periods and throughout the whole attack. An examination with the ophthalmoscope may reveal choroidal tubercles. There is a leucocytosis.

(7) **Catarrhal enteritis** in children, with prominent abdominal symptoms, may simulate typhoid fever very closely. In the former the symptoms are all gastro-intestinal save perhaps the occurrence of slight febrile disturbance and certain nervous phenomena, while typhoid fever manifests a wider range of symptoms (some of which are peculiarly its own—notably the greater prostration, more marked fever, enlargement of the spleen, and, above all, the characteristic eruption). In young children the last-named symptom may be either wanting or atypical, in which case the coexistence of enlargement of the spleen with other phenomena more or less characteristic of typhoid must suffice.

(8) **Salpingitis** on the right side may resemble typhoid. In the former there is usually a clear history either of antecedent vaginitis or of an abortion, and there exist special evidences of local peritonitis, with which may be associated the typhoid state, but not the classic features of typhoid fever. A digital examination *per vaginam* detects in salpingitis a tender mass occupying the right side of the pelvis, and the womb displaced to the left.

The diagnosis between typhoid fever and lobar pneumonia, with associated typhoid state, and appendicitis will be considered in the special discussion of these diseases.

**Prognosis.**—As in all other acute infectious diseases, so in typhoid, the prognosis depends upon three main considerations:

(1) **The severity of the type of the infection**, which is indicated in great measure, though not solely, by the degree of fever. A temperature of 106° F. (41.1° C.) is a serious symptom, and, if maintained at this point for a few days, an almost certainly mortal one. I have not seen a single instance in which the temperature has touched 106° F. (41.1° C.) for two or three successive days that has recovered. If the temperature mounts to and keeps at 105° F. (40.5° C.) for a longer period than three or four days, the case is also likely to prove hopeless, according to my experience. Temperatures above 106° F. (41.1° C.) I have not seen, and would regard their occurrence as offering no hope of



recovery. When the fastigium is prolonged, even though the fever be not exceptional, the prognosis is usually grave, while, on the other hand, marked nocturnal remissions show the course to be favorable. A sudden, deep fall, however, implies danger, and denotes intestinal hemorrhage, peritonitis, collapse, etc.

The researches of Isaac Ott have taught us not only that fever is due to an agent from within or without, which deranges the harmony of the thermotaxic, thermogenetic, and thermolytic apparatuses, increasing primarily tissue-metabolism, but also, that while high temperature is an indication of danger in specific fevers, it is not always the cause of it. He very properly regards high temperature as being only a part of an infectious process, and points out that the thermotaxic centers of the cortex may be so disordered as to alter the harmony between the heat-production and heat-dissipation. Under these circumstances a specific fever of severe form may be associated with a slight elevation of temperature.

The power of resistance to the influence of a greatly elevated temperature is quite reliably indicated by the condition of the heart. So long as the pulse is regular and its rate does not exceed 110 or 120 beats per minute, and provided the first sound of the heart is distinct, the outlook is favorable. When, however, the pulse maintains an average rate of 130 or more—a condition with which there is usually associated some degree of cyanosis, pulmonary congestion, and edema—the outcome is to be regarded as doubtful. Collapse is apt to follow the occurrence of sudden complications (perforation, hemorrhage, etc.), but it may also arise independently of such a cause. It is attended with grave danger for the patient.

Serious types are also shown by the occurrence of certain nervous symptoms, that may assume unusual gravity. This is particularly true of delirium, stupor, and the symptoms of motor irritation.

(2) **Circumstances of the Patient.**—Certain individual peculiarities render the prognosis highly unfavorable. It is *bad* in very *fat* persons. In such cases there is a great and constant danger of sudden collapse, and this fact also holds to a lesser degree with reference to those persons who are subjects of certain chronic diseases (Bright's disease, heart-disease, gout, emphysema, etc.).

**Age** is an influential modifying factor. After puberty the gravity of the disease increases with increasing years. After the fortieth year the relative death-rate augments much more speedily than prior to this period. Indeed, it may be said that, as a rule, typhoid has an unfavorable prognosis in persons past forty years, and chiefly for the reason that at this time of life there are dangers from an added liability to pulmonary complications and failure of cardiac reserve. In children (*vide* Clinical Varieties) the tendency to hemorrhage and peritonitis is reduced to a minimum, while the disease shows little tendency to assume a grave type. Hence in childhood typhoid gives the most favorable prognosis.

**The puerperal state** renders a typhoid patient liable to many accidents and peculiar complications, and it seems that independently of pregnancy the disease is more fatal among females than males. *Chronic alcoholism* is apt to be complicated with *delirium tremens*, often pre-

ceded by *pneumonia*, and to the latter disease the patient is very prone, perhaps to an equal extent with heart-degeneration and exhaustion.

The surroundings of the case affect materially the prognosis, poor sanitary conditions and poor attention greatly diminishing, and the opposite conditions greatly augmenting, the chances for recovery. Improved methods of treatment in recent years have also effected a decided lowering of the death-rate. Here it may be said that the average mortality of typhoid is from 8 to 10 per cent., as against 15 to 20 per cent. formerly. It must ever be remembered, however, that epidemics differ widely as to their mortality list—a fact which makes a precise statement regarding the question an impossibility.

(3) The third and last consideration is the presence or absence of dangerous complications and accidents. These have all been enumerated and their prognostic significance stated (*supra*). To merely reiterate some of those that lend fresh peril to the typhoid patient, arranging them with some regard for the order of their relative gravity, may prove helpful to the student. They are—perforation with diffuse peritonitis, intestinal hemorrhage, lobar pneumonia, lobular pneumonia, sudden collapse (due to cardiac weakness), excessive tympanites (often with marked diarrhea), and hypostatic congestion of the lungs. The fact that these complications and accidents of the disease are responsible, in a large measure, for many fatal results deserves especial emphasis.

### RELAPSES OF TYPHOID FEVER.

A relapse is a repetition of all the characteristics of typhoid after the latter has run its course. As a rule, the return occurs from one week to ten days after the beginning of convalescence, though it may be either earlier or later; and occasionally a relapse develops before the temperature has become normal, as occurred in 11 out of 21 cases recorded by F. C. Shattuck. The cause is a reinvasion of the blood by the typhoid bacilli or their secretions, but whether this is attributable to a reinfection from without or from within (most probably the latter) cannot be definitely stated. The pathologic lesions differ in no essential way from those described as belonging to the primary attack, but the stages through which they pass are not quite as long.

In the interval between the primary attack and the relapse there may be present suspicious features, such as a slight enlargement of the spleen, a trivial evening exacerbation of temperature, an unnatural apathy or dulness, and a more profound prostration than is usual. In the majority of instances, however, the relapse is announced by a reappearance of the characteristic febrile career, with an utter absence of any premonitory symptoms. The onset is rather more sudden than in primary typhoid. The temperature, however, rises in the characteristic "step-ladder" fashion, reaching the fastigium or second stage in two or three days, and the same relative abridgement of the fastigium and defervescence is observed. It follows that a relapse has a shorter duration than a primary attack, and, indeed, it rarely exceeds two to three weeks. It sometimes happens, however, that the temperature touches a higher limit in the relapse than in the primary attack, but, with rare exceptions, when the primary typhoid is of average or even a greater than average



severity, the temperature in the relapse does not reach an equal height. The characteristic rash appears earlier—from the second to the fourth day—and is somewhat darker and coarser than that of the first attack. The spleen swells rapidly and diarrhea often is present.

**Diagnosis.**—Upon the points that are distinctive of a primary attack of typhoid fever rests the important diagnosis between a relapse and a recrudescence. The latter is usually attributable either to errors in diet, to undue muscular exertion, or to great mental excitement; and, whilst it occurs during convalescence, it seldom lasts longer than one, two, or three days, and is not characterized by the diagnostic features of a relapse (peculiar temperature-curve, enlarged spleen, and specific eruption).

The **prognosis** of relapses depends very much upon the severity of the primary attack, those following severe attacks being relatively milder than those that follow the rudimentary, primary attacks.

The frequency of relapses differs widely in different epidemics. Hence the fact that the percentage of relapses as estimated by different authors ranges from 3 to 15 per cent. need excite no surprise. The relapse may repeat itself once, twice, or even thrice, and two relapses occur in about 1 per cent. of the cases. In a case which I<sup>1</sup> reported three successive and typical relapses occurred. The pale line or ridge which was mentioned (*vide* Clinical History) as noticeable in the nails after typhoid occurs similarly after each relapse, and in the afore-mentioned case of my own four distinct whitish, transverse ridges were perceptible after the conclusion of the third relapse.

**Recurrences.**—The term recurrence should be applied only to those instances in which successive attacks are separated by longer or shorter intervals after complete recovery from a previous or the primary attack. Typhoid fever usually bestows complete and lasting immunity against subsequent attacks, but this is not an invariable rule. Eichhorst has studied 600 cases, and found that in 28 of the number (4.7 per cent.) a second attack occurred. He also cites a case in which three or four attacks occurred in the same individual. I have seen two typical attacks of typhoid fever in two different persons, the intervals between the cases having been five and eight years respectively. Very rarely three separate attacks have occurred in the same person, and a second is usually milder than the first attack.

**Treatment.**—(a) **Prophylaxis.**—Modern hygienic resources happily enable us to minimize the number of occurrences of typhoid, and reports of the typhoid cases in a city may be taken as a safe indicator of its sanitary status. On the same principle it has been found that whatever tends to better the sanitary arrangements of a city tends in a decisive manner to diminish the prevalence of the disease, and this is particularly true of any improvements affecting the water-supply and drainage. Such facts as these show typhoid fever to be, to a large extent, a preventable affection.

The best means that can be employed during the attack, with a view to limiting the spread of typhoid, is *disinfection*, and the following brief description comprises its essential points as applied particularly to this disease:

<sup>1</sup> *Medical and Surgical Reporter*, vol. xlvii. p. 66.

**Disinfection** in typhoid may conveniently be divided into (a) that of the excreta (stools, urine, vomitus, and sputum); (b) of the bed and its coverings; (c) of the patient and the sick-room. While all of these subdivisions are of the greatest importance in the treatment of a case, *the disinfection of the excreta* (a) is perhaps most frequently overlooked or most carelessly performed, and hence the importance of the statement that all stools and urine voided by the patient, as well as all vomitus and sputa, should invariably be treated in the following manner: The excreta should be received in a vessel that can be thoroughly disinfected inside and out with any one of several standard solutions, of which that of chlorinated lime is the most effective and satisfactory. Bichlorid of mercury (1:500) also may be used, but, as it requires a much longer time and forms an insoluble compound with the albumins in the feces and sputa, it is inferior to the solution of chlorid of lime, which is now very generally used in a strength of six ounces per gallon of water. A 5 per cent. solution of carbolic acid is also much employed, and has proved of decided value.

It is my custom to order that one pint of the chlorinated lime solution be placed in the bed-pan (or other appropriate receptacle) *before* the discharges are received therein, and from one to two pints *after*. The whole is thoroughly mixed by stirring and shaking, care being taken that all solid masses are broken up. The vessel is then allowed to stand for three hours before it is emptied into the water-closet. If the mercuric-chlorid solution be employed, at least six hours must be allowed for thorough action upon the excreta. The urine must be treated in the same conscientious manner as the feces, vomitus, and sputum, and special care must be given to the latter two discharges in cases of pneumo-typhoid fever.

(b) It should be an invariable rule to change the bed- and body-linen daily, and as often as soiled. The mattress should be protected by a rubber cover, and this, together with the soiled linen and blankets, should be received in a sheet that has previously been dipped in a 5 per cent. solution of carbolic acid. The rubber sheets are to be washed with the carbolic-acid solution, but all other bed-clothes must be boiled for half an hour. When the patient leaves the sick-room the mattresses are to be fumigated and aired daily for a week, and the rubber covers and bedsteads washed with a solution of mercuric chlorid (1:1000).

(c) After every stool the patient must be thoroughly cleansed with a compress of cloth or cotton wet with a solution of mercuric chlorid (1:2000) or of carbolic acid (1:40). The bed-pan and hopper are to be treated in the same manner, and the cloths used are to be immediately burned. Fitz recommends that the feeding utensils be cleansed in boiling water immediately after using. It is very important that the sick-room should have thorough daily ventilation, and with this end in view it is of advantage to devote adjoining rooms to the use of the patient, in order that the windows may be opened in the one not occupied by him, and the other ventilated through it.

Finally, since it is well known that many epidemics are directly traceable to the drinking-supply of water and milk, it is necessary that all water and milk used by the patient and other members of the household be boiled for half an hour before being ingested, and if an epi-



demic be prevailing, the whole community should join in this valuable precaution.

The municipal authorities possess in thorough filtration a power that might and should be used to advantage, as has been well shown by the improvement effected in certain water-supply and sewer systems—*e. g.* in Vienna, where by purification of the water-supply the death-rate in typhoid fever was reduced from 12.5 per 10,000 to 1.1 per 10,000.

**Isolation of Patients.**—I am of the firm belief that it is advisable to isolate typhoid cases as far as possible—*e. g.* in hospitals, to keep them in special wards; in private families, in special apartments, into which none but the attending physician and the nurse should enter. There is incontestable proof that typhoid fever is feebly contagious,<sup>1</sup> and, moreover, that it obeys the laws of contagious diseases. The preventive treatment must be conducted on the same principles as those governing the treatment of other infectious diseases, and it must aim to cut off not only the chief, but every channel of communication from the sick to the healthy. The demands of hygiene cannot, under less exacting conditions, be fully satisfied.

**Prophylactic Inoculations.**—Encouraging results have also followed the *preventive inoculation* of healthy persons with typhoid virus, and Pfeiffer believes that this mode of prophylactic treatment promises to render great service in future epidemics. Iwanon has successfully immunized monkeys, and Pfeiffer has had considerable success in inoculating human subjects with the virus.

(b) **Treatment of the Attack.**—(1) The **general conduct** of the case, including skilful nursing, is of paramount importance to the typhoid patient. He should be put to bed as soon as the indications point to this disease, and kept there continuously in the recumbent posture till the end of the attack. The sick-room should have a sunny exposure if possible; should be cool and well ventilated, though free from strong currents; and perfect cleanliness both of the room and of the utensils employed in the management of the case should be attempted. The bed should be provided with a woven-wire mattress, upon which should be placed one of hair. A rubber cloth is spread beneath the sheet, and the latter kept smooth in order to lessen the danger from bed-sores. A seriously ill patient should lie on an air-cushion or, better still, a water-bed, and to avoid bed-sores he should be instructed to turn gently to either side from time to time. His back, hips, and heels should be bathed frequently with a mixture of alum and salt in dilute alcohol. The use of the bed-pan and urinal is an absolute necessity. When a good nurse cannot be had, the attending physician must note *in writing* the directions regarding the disinfection of the excreta, bed-linen, and utensils, as well as regarding the exhibition of the food, medicine, etc. The mouth and throat should be kept clean, since by so doing we obviate unpleasant and even dangerous complications (apthous ulcer, thrush, parotitis, lobular pneumonia, etc.), and in mild cases the patient himself can attend to this, though not in severe attacks. In the latter the nurse or attendant should wash the mouth and tongue several times daily with a solution of boric acid (3 per cent.), and the throat may be sprayed at equal intervals with a similar solution. A frequent moistening of the

<sup>1</sup> For illustrative cases see *Philadelphia Hospital Report*, 1891, vol. i. p. 149.

tongue and mouth, and particularly the lips, with glycerin and water (equal parts) gives great comfort when they are dry and parched.

(2) An **appropriate liquid diet** is highly serviceable, and the best article of food is milk, which it is well to dilute with plain water (or lime-water), since aerated waters are objectionable in that they sometimes increase the meteorism. The daily quantity should not be less than three pints, and it is important that the stools be examined, since, if the milk be not thoroughly transformed, curds or (on microscopic examination) numerous fat-globules will be seen, in which case a smaller amount should be given. If curds or fat are still seen, the milk should be peptonized. Experience teaches that milk is often better taken and better borne when a little brandy, coffee, or tea is added to it. When milk cannot be taken or digested in sufficient amount, either whey, sour milk, or buttermilk may be tried; and if these be distasteful, we may replace them (wholly or in part) by meat-juices or broths of various sorts, together with one of the standard infant's foods made with milk or water. Albumin-water, prepared by straining egg-white through a cloth and adding an equal part of water, has given much satisfaction in my hands. It may be made pleasant to the taste by flavoring with vanilla or lemon, and with meat-juice and broths will often support a patient during the most trying period of the attack. There are typhoid subjects who cannot (on account of vomiting, etc.) take *per oram* sufficient nourishment to support life. In such cases we may substitute or supplement the usual method of feeding by rectal alimentation, when from 3 to 4 ounces (96.0–128.0) of peptonized milk,  $\frac{1}{2}$  ounce (16.0) of meat-juice, and a little egg-white may be combined, and employed at intervals of four hours. In the case referred to previously, in which large hemorrhages occurred, vomiting was a distressing symptom, and the patient was nourished at times in part, and then altogether, by *nutrient enemata*. The latter mode of feeding in this disease may be the means of saving life, and it deserves a more extended trial than it has hitherto received at the hands of the profession. In early convalescence the patient may take well-cooked plain rice, entire eggs (diluted), or thin custards. Solid food should not be allowed till the temperature has been at the normal grade for one week at least. When defervescence is much prolonged and the patient becomes very weak, the administration of soft food (eggs, finely scraped meat, etc.) is often followed by notable improvement and is a justifiable procedure. The return to a full, solid diet should invariably be very gradual.

Pure cold water is an excellent drink for fever patients, and it should be offered to them regularly when on account of hebetude, delirium, or coma they do not ask for it. Coffee, tea, lemonade, and similar beverages, sweetened with glycerin or saccharin, are admissible.

(3) **Stimulants** are useful in most cases, though by no means in all; but when the heart becomes enfeebled alcohol should be used regardless of the temperature. In severe types whiskey is the best form: in milder ones, some good wine, such as port, sherry, or madeira. It is well to begin with a moderate daily quantity, and then increase, if necessary, until the indication is fulfilled. If the patient so desires, we may use brandy instead of whiskey, and it is usually toward the close



of the second or during the third week of the disease that the indications for the use of alcohol arise. It is not only the best spur for a flagging heart, but is of equal value in combating unfavorable nervous symptoms; and the time for commencing its use may be indicated first by the latter symptoms (*e. g.* delirium, coma, tremor, twitchings, etc.). The quantity to be administered must be regulated by its effects, which must be carefully studied in every instance, since it may act injuriously, and even aggravate the symptoms, though this is seldom the case. Threatened collapse may be met by full doses of alcohol ( $\frac{1}{2}$  ounce (16.0) every hour), which should be supplemented by strychnin (gr.  $\frac{1}{15}$ ; 0.004 every three hours), exhibited subcutaneously till the depression has been in great measure counteracted, and then continued in medium-sized doses (gr.  $\frac{1}{30}$ ; 0.002 every four hours). Other cardiac stimulants are worthy of trial for their favorable supplementary action, and among these are digitalis, sulphuric ether, etc.

(4) **Hydrotherapy.**—There is at the present day general agreement among medical authors that the best mode of treating typhoid fever is by means of the *cold bath*, which was originally introduced by Currie of London (more than a century ago), and reintroduced and successfully practiced by Brand of Stettin. There are obstacles in the way of carrying out hydrotherapy in private families, but since convenient and inexpensive portable tubs have been devised by Batt and Furbush of Philadelphia, Burr of Chicago, and others, most of the valid objections to the method have been removed. At all events, the benefits offered to the patient by this method are so great and varied that it becomes the duty of every physician who treats typhoid fever to be prepared to employ it. The beneficial influences of the baths are as follows: (1) They absorb the body-heat directly, thus reducing the temperature and overcoming the ill effects of high fever, this action becoming more marked after a day or two of the treatment; (2) They improve the nervous symptoms, also render the mind clear; they diminish mental dullness, stupor, muscular tremors, and twitchings, and induce sleep; (3) They strengthen the heart, thus obviating the danger of sudden circulatory collapse and the consequences of increasing cardiac weakness (hypostatic congestion of the lungs, venous thrombosis, etc.); (4) They stimulate the respirations, whereby the inspirations are deepened and the tendency to pulmonary complications greatly lessened, especially severe bronchitis, lobular pneumonia, etc.; (5) The renal function is invigorated, and as a result the elimination of typhotoxins by the kidneys is increased (Roque and Weil); (6) On account of the cleanliness of the skin which they ensure, bed-sores rarely occur; (7) They may shorten the stay in the hospital or sick-room, but not the stay in bed, except, perhaps, in the lighter types.

Unquestionably, the good effects of the Brand method receive striking confirmation from statistical reports which have been prepared by Brand himself, Jürgensen, and others abroad, and by Baruch, Osler, Wilson, and others at home. According to the warmest European advocates of the method, the mortality is less than 0.5 per cent., and no deaths occur in cases that come under treatment before the fifth day. The results among American clinicians, however, have been less flattering, though strikingly uniform, and show an average mortality of 7.3

per cent. Of 102 cases of my own treated by cold and by graduated cold baths, 8 died—giving a death-rate of 7.8 per cent.—while prior to the use of the cold baths the lowest mortality was between 13 and 15 per cent.

The *details*, which we will now consider, connected with the administration of this plan of treatment are of the utmost importance. The tub is to be brought to the bedside of the patient, and in hospital practice both bed and tub should be screened while the bath is in progress. After removing the night-dress and placing a large napkin around the loins, the patient should be lowered into the bath by a sheet held at each corner by an attendant (and, if seriously ill, with the least possible disturbance), and there carefully supported and held while in the bath. If sleeping, the patient must be awakened and the bath delayed for ten or fifteen minutes. Young subjects and adults in light cases of the disease may be handled properly by two persons, but I do not approve of allowing the patient to step from the bed into the bath, however light the case. While in the bath the skin-surface, particularly that of the back and limbs, is constantly rubbed by the attendants, in order to stimulate the peripheral circulation and as far as possible to avert chilliness and discomfort. The head of the patient rests upon a rubber air-cushion. At first he should be kept in the bath five to eight minutes; later, ten or fifteen minutes, according to the severity of the case. The head and face are bathed at once from a basin, and a cold compress is applied to the forehead, and, if prominent nervous symptoms be present, often associated with high temperature, water at 70° F. (21.1° C.) or lower should be poured from an elevation of about six inches upon the head and nape of the neck several times during the bath. The ears must be stopped with cotton when douching is practiced. If while in the water the patient complains bitterly of the cold or is very restless, a stimulant may be administered—f5j (32.0) of whiskey, diluted—and if this fails he must be lifted into bed and further stimulated. If he be very young, highly sensitive, or elderly, it is best to place him at the commencement in water of a temperature of 85° or 90° F. (29.4°–32.2° C.), and then gradually cool it down to 80° F. (26.6° C.). After he has become accustomed to the bath he may be immersed in water at the temperature of 80° F. (26.6° C.), to be reduced to 75° F. (23.8° C.) or even 70° F. (21.1° C.), below which it is unnecessary to go save in the rarest instances. This is the *gradually cooled bath* of Ziemssen. In the rigid Brand method, which is now generally adopted, and which I employ except in the above-mentioned instances, the patient is lifted at once into a bath at 70° F. (21.1° C.) and kept there for fifteen minutes. The patient is to be removed from the bath to the bed (previously protected by a blanket and mackintosh), wiped off gently, after which the sheet, blanket, etc. are withdrawn and he is covered with a blanket. If now reaction be retarded, some hot broth or about an ounce of whiskey should be administered and active friction applied to the back and extremities.

The effect of the bath is best shown by the rectal temperature, which is taken half an hour after the conclusion of the bath, and again a half hour later if the patient be not asleep. Usually the temperature will be found to be two or three degrees lower than before the plunge



Case No. ....

**DIAGNOSIS**

Typhoid Fever

Revise .....

**Notes of Case**

Name Boela Romans M.F.

Age 28 Years

Nativity N. B. A.

Occupation Operator

Residence 216 Union St.

Philada., Pa.

Date of admission 9-14-95

**Diet**

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**Treatment**

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Result .....

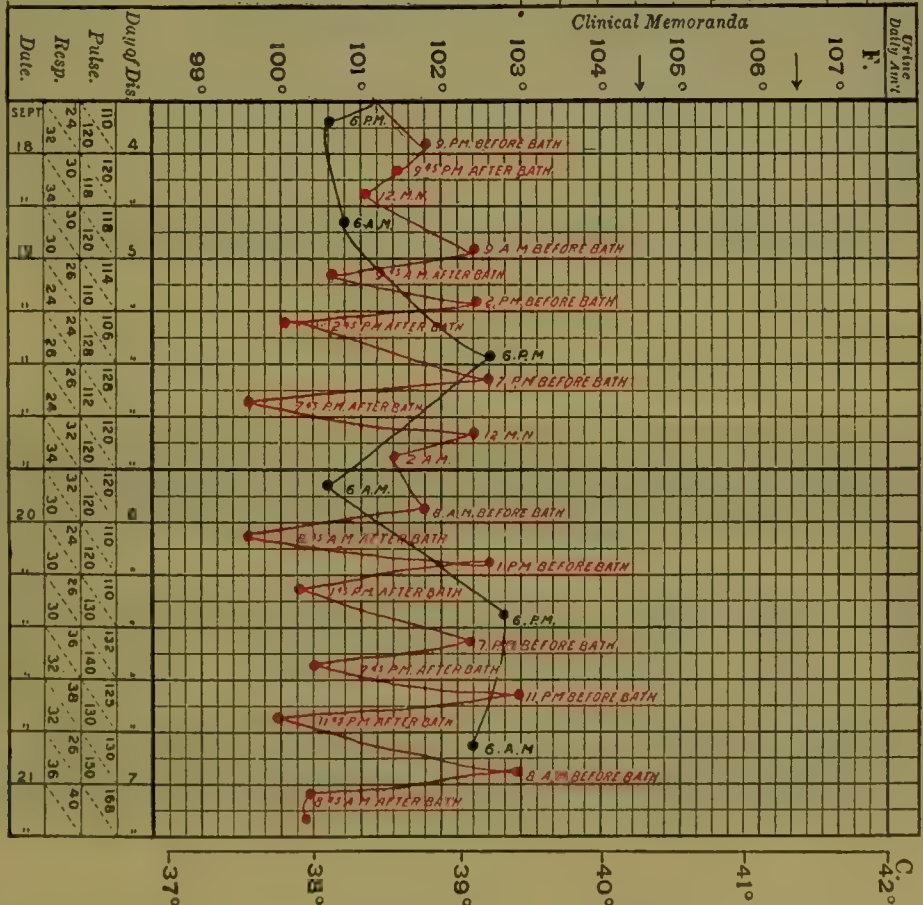


FIG. 5.—Chart illustrating the influence of cold baths in the treatment of typhoid fever.

(see Fig. 5). In obstinate and severe cases the fall may be less than one degree, in which case it is advisable to either prolong the bath to twenty minutes or to reduce still further the temperature of the water. Protracted warm baths are highly recommended by Reisse and others when cold baths are badly borne or are unproductive of good results.

In light cases the cold bath should be repeated every six or eight hours; in severe ones, every three or four hours, but more frequently than once in three hours is not advisable, even in the worst cases. Sufficient water to immerse the patient to the neck (about 30 gallons—114 liters) should be used. During the night the patient should be allowed to sleep for six or eight hours if he can do so.

As before stated, there are a number of convenient and satisfactory portable tubs in the market, but that devised by Dr. C. L. Furbush of



FIG. 6.—Portable bath-tub in use.

Philadelphia possesses certain leading advantages (Figs. 6, 7). The frame is made of light wood, and when folded is 4 inches (10.156 cm.) in



FIG. 7.—Portable bath-tub, folded.

depth, 14 inches (35.546 cm.) in width, and 5 feet 10 inches (1.778 m.) in length, so that it can be placed in a closet or beneath a bed. Less than two minutes are required to prepare the bath, which the patient receives while lying in bed. When in use the ends are fastened by brass pins hung on small chains, and these hold the frame in a fixed position. The tub proper is made of double-faced sheeting, reinforced in the middle, so as to resist the greatest amount of pressure. The



sides of the sheet have a casing through which is passed a wooden rod 4 feet 4 inches long (1.320 meters), and outside of this a margin of  $1\frac{1}{2}$  inches (3.808 cm.) is left for the brass eyelets, through which passes a rubber cord which is covered with woven cotton. This cord, which is attached to the sheet, is held to the frame by special brass fittings along the lower sides of the latter. By the use of the cord and wooden rods we have an even tension on both sides, combined with ample resistance to withstand the pressure of the water. An adjustable head-rest fits into the end of the frame. The wooden rod also enables the attendant to roll up the sheet quickly after the bath. Through the bottom of the sheet a 1-inch rubber tube is fitted with a stopper, and by means of this the tub can be emptied much sooner than by a siphon. The frame is covered with ivory-enamel paint, and can be cleansed easily, and the entire weight of the outfit is 25 pounds (11.33 kgs.).

Whenever practicable, and when contraindications do not exist, baths are to be instituted and carried forward religiously, no matter how mild the case promises to be at the onset. Brand recommends that the baths be commenced when the temperature in the rectum reaches  $102.2^{\circ}$  F. ( $39^{\circ}$  C.). The height of the temperature, *per se*, is not to be regarded as an absolute indication for the employment of the cold bath, since the facts must ever be remembered that the essential effect is a stimulation of the nerve-centers which preside over the organic functions, respiration, circulation, etc., and that reduction of the temperature is the secondary effect. Moreover, cold baths exert a marked preventive effect in obviating serious symptoms and complications. I continue the baths until the evening temperature remains below  $101^{\circ}$  F. ( $38.3^{\circ}$  C.).

The *contraindications* to the use of baths are—(1) *Intestinal hemorrhage*, which is in itself attended with danger and requires absolute quiet for a time (four days), when the baths may be resumed if there is no recurrence. (2) *Peritonitis*, the occurrence of which always excites suspicion of perforation. Here, again, rest and all that the term implies must be procured. (3) *Extreme Cardiac Weakness*.—The excitement in the necessary handling of the patient connected with the bath might prove fatal, as I have witnessed in one instance. This condition is sometimes met with in cases that come under observation at a late period, and have not been brought under proper treatment from the start, or in cases arising in aged and previously enfeebled subjects. (4) Cases that have progressed to an advanced stage (the third week of the disease) should not be immersed. Dangerous and even fatal collapse has been observed to follow cold baths under these circumstances.

*Substitutes for the Cold Bath*.—The prejudice which exists against the cold-bath treatment—at least in America—sometimes proves insurmountable. Again, there are many physicians who do not avail themselves of the means at command for carrying out hydrotherapy. In consequence of these facts substitutes for the cold and the gradually cooled baths are, unfortunately, quite commonly in vogue. Among them cold sponging of the body of the patient is often resorted to, though it secures for him few and trivial advantages as compared with those of the baths. If this method be employed, the water should be of the temperature of the air of the room or ward. The limbs should

be sponged and dried in succession, and then the trunk. Whenever the temperature reaches  $102.5^{\circ}$  F. ( $39.1^{\circ}$  C.) this measure is to be instituted, each sponging being continued until the desired effect has been produced (a reduction of the temperature of  $1\frac{1}{2}^{\circ}$  to  $2^{\circ}$  F. or  $1^{\circ}$  C.), unless the patient gives signs of uneasiness, when it must be cut short. It may be repeated as often as required. To the water used for the applications equal parts of vinegar or spirits should be added. The efficacy of the cool sponging is greatly enhanced by the simultaneous application of the ice-cap, either constantly or during alternate hours.

If this method fails, as it often does in severe types, the cold pack may form a satisfactory substitute; and I have found it of great use with children, in whom the reaction after a cold bath is often delayed or imperfect. The patient is placed upon a cot previously prepared by spreading over it a blanket, which is in turn covered with a sheet doubled and wrung out of water of the required temperature,  $70^{\circ}$  to  $80^{\circ}$  F. ( $21.1^{\circ}$ – $26.6^{\circ}$  C.). The sheet and blanket are now wrapped about the patient evenly, and he is left in the pack for a period varying from a half to one hour. Free diaphoresis generally ensues, and this aids in maintaining the fall of temperature. The effect, in most instances, is to reduce the body-heat two degrees or more, and the treatment may be repeated at intervals of three or four hours if needful. The wet sheet alone may surround the patient, and be sprinkled at short intervals with a watering-pot containing water of a temperature of  $70^{\circ}$  F. ( $21.1^{\circ}$  C.). In desperate cases in which cold baths are for adequate reasons out of the question ice-water enemata may be tried. If carefully administered, they accomplish a reduction of the temperature sometimes by two or more degrees. Leiter's coils may be applied to the head, chest, or abdomen, and form the most convenient method of applying cold.

Guaiacol has been used for its potent antipyretic effect by H. G. McCormick and others, from 10 to 30 minims (0.666–2.0) being applied to the skin surface. I have seen its use followed by rigors, hyperpyrexia, etc., but McCormick has adopted the rule of using sufficient only to lower the temperature to  $100^{\circ}$  F. ( $37.7^{\circ}$  C.), and has thus avoided all ill effects.

(5) **Internal Antipyretics.**—Internal antipyretics are also effectual agents in combating immoderate temperatures, and certain of them, it must be confessed, have a powerful influence. Moreover, they soothe and moderate the nervous symptoms and act more or less potently as antiseptics. But even the most reliable of this group of medicaments (phenacetin, acetanilid, and antipyrin) are open to the serious objection that they depress cardiac power, and on account of this I do not use them in hospital practice (where the Brand method can be rigidly carried out), and very rarely indeed at any time in private practice.

When the Brand method cannot be employed, or, as rarely happens, it is ineffective and there is present a high fever with decided nervous symptoms, internal antipyretics are allowable if properly administered. The safest among them is phenacetin, of which 5 grains (0.324) may be given at a dose (preferably about 3 P. M.), and repeated after four hours should the first dose fail of the desired effect. Acetanilid is more effective than phenacetin, but is not quite so free from injurious action as the latter. It may be prescribed in doses of 2 to 4 grains (0.129–



0.259), to be followed by a second dose of equal size in four hours if necessary. The heart is always to be guarded by the use of stimulants when internal antipyretics are exhibited.

(6) **Intestinal antiseptics** are much used to destroy the bacillus of Eberth or to counteract the ill effects of its toxins. Unquestionably they meet neither of these leading indications, but they are called for in an affection in which extensive intestinal ulceration and moderate tympanites are usual manifestations. The bowel antiseptic which I have employed quite extensively, and with uniformly good results, is salol, this drug being broken in the intestinal canal into carbolic and salicylic acids, and being capable of controlling meteorism as nothing else has done in my hands. The dose is 2 to 3 grains (0.1296–0.1944) every three hours, preferably administered in capsule. With it I usually combine quinin in doses of 1 to 2 grains (0.0648–0.1296) each. Henry speaks strongly in favor of thymol, which he prescribes in pill or capsule (gr. iiss–0.1620) every three or four hours. I have employed this agent recently, and with gratifying results, in a few cases in which debility was a prominent feature.

*Lactophenin* (gr. vij–xv—0.4536–0.9720 per dose), in starch capsules, up to  $1\frac{1}{2}$  drams (6.0) daily, according to the indications, is highly recommended (Jaksch). Carbolic acid, iodine, and other antiseptic agents have their advocates, but my own experience with them has been too limited to warrant an expression of opinion as to their value in this disease.

*Turpentine* fulfils in some cases a leading indication. When the tongue is dry and brown, the abdomen distended, the general prostration marked, and often muttering delirium present—symptoms of the typhoid state—the use of this agent, together with alcoholics, constitutes the best mode of treatment. Turpentine is best given in a capsule in the form of white turpentine—dose, 3 to 5 grains (0.1944–0.3240) every three hours. Its routine administration, however, is to be unqualifiedly condemned.

(7) **Curative Inoculations with Cultures of Serum.**—The brilliant results obtained from the use of antitoxic serum in diphtheria and certain other affections have led to attempts at curative inoculations in typhoid fever. Though their specific virtue is yet to be demonstrated, it is deemed proper to state the results which have been obtained as concisely as possible. E. Fränkel and Manchot have treated 57 cases of typhoid fever with a sterilized liquid derived from a culture of the bacillus of Eberth in thymus bouillon and heated to 140° F. (60° C). Of this,  $\frac{1}{2}$  c.cm. was injected deeply into the gluteal region. No reaction followed the first injection. The next day 1 c.cm. was introduced into the other buttock. This produced an elevation of temperature with chilliness, followed in three or four hours by decided sinking of the temperature. The fever, however, rose again if the injections were now omitted. Moreover, when the latter were continued at intervals of two days in augmenting doses (1 c.cm. each day), the fever assumed the remittent type and disappeared altogether after a few days. The splenic enlargement and roseate spots, however, persisted.

Rumpf, following the methods of Fränkel as to preparation and administration, treated 30 cases of enteric fever with cultures of the bacil-

lus pyocyaneus, with like results. F. Kraus and Bushwell, after treating 12 cases with the sterilized pyocyaneus bouillon, however, concluded that this method possessed no specific curative value.

Hughes and Carter treated a number of cases with blood-serum derived from convalescent cases, but apart from a decided lowering of temperature the general course of the disease was not perceptibly modified. More recently, Klemperer and Levy have obtained the blood-serum from dogs (after inoculating them with bouillon-cultures of typhoid bacilli), and found it to be capable of immunizing susceptible animals, as the guinea-pig, etc., against the action of typhoid bacilli, and also of curing them when infected. This treatment was employed in 5 cases of human typhoid, all of which pursued a mild course.

Quite recently Pfeiffer and Kolle have shown the presence of a bactericidal substance in the serum of enteric-fever patients, as well as in that of certain immunized animals. but the result of their important researches has furnished a means of diagnosis of the disease rather than a specific means of cure (*vide* Diagnosis).

(8) **Treatment of Individual Symptoms and Complications.**—*Headache.*—Early in typhoid the headache demands relief. Absolute rest and cold to the head frequently suffice. Depressant analgesics are to be avoided so far as may be, though it sometimes becomes necessary to resort to them. At such times those least objectionable are to be selected. I have found that a mixture containing sodium bromid (gr. x to xv—0.6480 to 0.9720) and the deodorized tincture of opium (℥iij to v—0.1998 to 0.3330) in each dose, given at intervals of three or four hours, exercises a striking palliative influence. In occasional instances the above mixture fails, and then phenacetin (gr. ij to iij—0.1296 to 0.1944) may be substituted for the opium in the same combination or separately in capsule.

*Insomnia.*—The cold baths or other measures calculated to relieve the headache often procure for the patient refreshing sleep. It is important not to allow him to go too long without sleep, since this tends to the development of a pronounced “typhoid state” and its concomitants. When the agents recommended for the headache fail, I employ morphin hypodermically in small doses (gr.  $\frac{1}{16}$  to  $\frac{1}{8}$ —0.004 to 0.008) during the evening hours, with excellent results, and have yet to witness the unpleasant after-effects or the unfavorable influence upon the secretions that have been described by some authors. Codein, sulfonal, and, more recently, chloralamid, have proved useful.

Chloral is more certain in its action than the above agents, but I have abandoned its use for the reason that it apparently produced circulatory collapse in two instances.

*Delirium.*—Since the introduction of the Brand method delirium rarely calls for special medication. I have observed, in common with others, particularly during the advanced stages, that in cases in which the circulation was feeble and in which typhomania was a prominent feature, the administration of stimulants with a free hand completely dispelled the nervous phenomena. If alcohol fails, ether (℥x—0.666—at a dose) may be given hypodermically, and repeated in one or two hours if necessary. To combine with the arterial some nervous stimulant (musk, valerian, etc.) will be found serviceable, particularly in cases



in which the delirium assumes an hysteric type. Of special value in meeting this symptom are the bromids, hyoscyamus, the persistent use of ice to the head, and the other agents suggested for the headache and insomnia.

*Vomiting* is rarely troublesome. Its chief cause is the irritation of the gastric mucosa, which may be caused by improper diet or medication. The best measure for the relief of this symptom, after the removal of the cause, is the use of ice, taken in small pieces and swallowed. If vomiting occur during the period of development, minute doses of calomel, combined with sodium bicarbonate, may be prescribed with good effect. If it occur during the fastigium, the amount of milk taken should be reduced by one half, peptonized, and then diluted, preferably with lime-water. If the patient experience a strong aversion to milk, it must be suspended temporarily and liquid beef-peptonoids or broths substituted. Dry champagne may be administered simultaneously. Excessive irritability of the stomach calls for perfect rest of the organ for a period of not less than twenty-four hours, the patient being meanwhile supported by rectal alimentation and subcutaneous medication.

*Diarrhea* more than any other single symptom claims special attention. Two to four movements daily do not constitute diarrhea and do not demand treatment, but if this number of stools be exceeded, the condition should receive consideration. It may be caused by overfeeding or by improper food—as shown by the stools, as a rule—in which case regulation of the diet is curative. It is often due to ulcerated and catarrhal lesions of the intestines, and particularly the large bowel, and in such cases requires medical interference. Unquestionably, the use of proper intestinal antiseptics and such as possess the property of insolubility to a high degree is most valuable. Astringents may be combined with the latter or given separately. The subjoined formulæ have yielded better results in my own hands than numerous others which have been tried:

R<sub>y</sub>. Bismuth. salicylat.,    ʒij (8.0);  
       Betanaphtol,            ʒj (4.0).

M. et ft. capsulæ No. xxiv.

Sig. One to be taken every three hours.

Or,

R<sub>y</sub>. Salol.,                    ʒj (4.0);  
       Bismuth. subgallat.,   ʒij (8.0).

M. et ft. capsulæ No. xxiv.

Sig. One every two or three hours.

Or,

R<sub>y</sub>. Plumbi acetat.,        gr. xxiv (1.555);  
       Ext. opii,              gr. iss-ij (0.097-0.1296).

M. et ft. pil. No. xij.

Sig. One every three or four hours, as required.

The last formula may be administered in the form of a suppository, both ingredients being doubled in quantity.

Late in typhoid fever, when the ulcers are fully developed, opium is

the remedy *par excellence*, since it tends to arrest the peristaltic action which keeps up the diarrhea and favors the spread of the inflammation to the peritoneum. I have recently observed brilliant results from the use of rectal injections of an astringent solution (tannic acid 1-2 per cent.), alternated with an antiseptic solution (salicylic acid 1-2 per cent.), each given once daily at intervals of twelve hours.

*Constipation*, which is often present, and particularly until the middle of the second week, is to be relieved by simple enemata of soapsuds every second day. Calomel may be used in the early stage of dynamic cases. Its employment in this manner may be followed by symptoms of a milder type than are ordinarily encountered. If constipation exists during the third week, accompanied by an oscillating temperature-curve, as rarely occurs, saline laxatives in small but repeated doses may cut short the attack.

*Tympanites*.—This is sometimes a most distressing symptom, and is often associated with marked diarrhea. The claim has been made that if turpentine be administered in suitable doses throughout, both tympanites and diarrhea are controlled. Turpentine is a good remedy, but only when certain indications exist (*vide supra*), and it is without the power to influence the general course of the affection. As a remedy for tympanites it is excellent and richly deserves a trial. When employed for this symptom alone I prefer to apply it in the form of stupes over the abdomen, although when, as is frequently the case, the gases occupy chiefly the large bowel, turpentine enemata should be given, and, these failing, a long rectal tube should be passed.

The meteorism is often increased by the milk taken, and a change of food from the latter to liquid peptonoids, meat-juices, and albumin-water cures some and helps others.

*Hemorrhages* from the bowels, however slight, demand prompt and close attention, and complete rest must be secured immediately. The bowel-movements, if the hemorrhage has been copious, must be allowed to pass into the draw-sheet. The ice-bag (suspended if possible) should be applied to the right iliac region, and ice freely given by the mouth. Opium, to control peristalsis, is our chief reliance among medicinal substances. It should be administered in small doses at frequent intervals and, by preference, hypodermically. It may be combined with full doses of the acetate of lead to arrest the bowel-movements. Cases in which slight oozing appears from time to time are best controlled by the latter combination in pill form. In similar instances turpentine is quite efficacious, and it is also warmly recommended for copious hemorrhages by many authors. Ergotin may be used (hypodermically, to be repeated every hour) in severe bleedings. The amount of food should be greatly restricted for about twelve hours.

*Peritonitis*.—When this complication is due to perforation of the intestine the patient in almost every instance passes quickly beyond hope, though recovery does rarely take place when nature, with or without the aid of the physician, limits the inflammation by the formation of adhesions. Morphin should be given hypodermically to relieve suffering, and the laparotomist should be called immediately. Operation offers little hope of cure, on account of the previous unfavorable local and general condition, but with the progress of convalescence the



chances of recovery from this accident improve. Peritonitis due to direct extension of the infectious inflammation of the bowel without perforation often admits of successful treatment. Unless perforation be suspected the physician is justified in administering saline purgatives, at the same time controlling pain by means of small doses of morphin, which is without harmful effects, save that it somewhat delays the recovery.

*Pneumonia.*—*Broncho-pneumonia* was formerly (under the old régime) the most frequent pulmonary complication, and is to be treated in the manner indicated in the section on this affection.

*Lobar Pneumonia.*—The treatment of that form of pneumonia which occurs in the advanced stage of typhoid will be considered hereafter (*vide* Secondary Pneumonia). That variety of lobar pneumonia which rarely inaugurates typhoid requires the same treatment, until the true typhoid symptoms arise, as adynamic forms of lobar pneumonia (*vide* p. 154).

The *hypostatic congestion* of the bases of the lungs due to cardiac weakness and the decubitus of the patient is to be met by heart-stimulants and by changing the position of the patient.

*Bronchitis.*—No special measures are necessary when the bronchitis is confined to the larger tubes, as in typical cases, while, if severe and diffuse, its management is identical with that of broncho-pneumonia, to which it leads.

*Laryngitis.*—For this condition, which rarely develops in typhoid fever, counter-irritation should be tried, and if this brings no relief a small blister may be applied below the angle of the jaw on either side. For edema of the larynx scarification and the inhalation of simple or medicated steam are measures to be used. Then, should suffocation become imminent, tracheotomy should be performed without delay.

*Bed-sores.*—The preventive measures have already been considered, but the smallest bed-sore demands active treatment. It is to be kept clean by means of a weak solution of some antiseptic, and may then be dusted with a powder composed of equal parts of boric acid, calomel, and bismuth; if sluggish, with a powder made up of aristol and iodoform. I have found unguentum balsami peruviani (1:30) to be a valuable remedy in bed-sores. Should the edges of the ulcer become undermined, a drainage-tube is sometimes necessary.

*Thrombosis of the femoral vein* is best treated by elevating the part and keeping it at perfect rest. The following ointment may also be applied along the course of the vessel:

R $\bar{y}$ . Ung. ichthyol.,

Lanolin,

Ung. belladonnæ,

$\bar{a}\bar{a}$ .  $\bar{z}$ j (8.0);

q. s. ad  $\bar{z}$ j (32.0).

Sig. Apply three times daily.

After the swelling has subsided an elastic stocking should be worn for a couple of months.

(9) **Management of Convalescence.**—Some of the points connected with this subject have already been discussed (*diet, time for getting up, etc.*). I may add that should a recrudescence occur the patient should be kept at rest in the recumbent posture and a return made to the liquid forms

of food. Often a moderate laxative serves a good purpose, particularly if an indiscretion in diet have been committed. The ulcers may not be healed, though the temperature may have been normal for a week or ten days; hence the patient should not be allowed to stir about for a period of two weeks after the temperature has returned to the normal. At first his movements should be slow; he may soon, however, be allowed to exercise gently in the open air during seasons of favorable weather. Mental excitement is to be avoided, since it may produce a recrudescence of fever. Occasionally, during convalescence the diarrhea persists, being due to colonic ulceration, and is best treated by restricting the diet to milk and other light forms of albuminous food. The patient must be confined to bed. Medicinal treatment by the oxid of zinc internally and the use of astringent and antiseptic rectal injections, as before indicated, usually proves successful. Constipation may be a troublesome symptom in convalescence, and is best relieved by simple enemata. Most patients require tonics. We should begin with a vegetable salt of iron in combination with a simple bitter (such as the infusion of gentian), and later an inorganic salt of iron, with quinin and strychnin, may be resorted to. If there be a predisposition to tuberculosis, cod-liver oil and creasote should be given for a period of two or three months. Relapses are to be treated as primary attacks, and recurrences in the same manner.

#### MOUNTAIN FEVER.

The term "mountain fever" should be regarded as applicable only to that condition which develops shortly after ascent to a very high altitude. There is no definite *pathology* nor *etiology*, but the symptoms are attributable to the effects of a rarefied air upon the organic functions (respiration, circulation, etc.).

The **symptoms** are a much quickened pulse, urgent dyspnea, headache, vertigo, and at times nausea and vomiting. There is a subfebrile movement, the temperature touching 100° or even 101° F. (38.3° C.). Thirst is present and the appetite is lost. Malaise and a sense of exhaustion on attempting exertion are experienced. Hemoptysis has been noted, but rarely. The effect upon the human economy of high altitude varies with the extent of the differences in individual reserve nerve-force. Rest and acclimatization will almost invariably restore healthy function.

Different clinical observers have depicted as mountain fever various forms of illness which might have been as properly referred to other well-recognized diseases, especially *typhoid fever*. The lesions of typhoid fever were present in two instances that were necropsied. Curtin, however, has reported four cases all evincing the signs and symptoms of lobar pneumonia. It must not be forgotten that high altitude may alter the clinical peculiarities of the acute infectious diseases.



## TYPHUS FEVER.

(Ship-fever, Camp-fever, Jail-fever, etc.)

**Definition.**—An acute contagious disease of unknown specific etiology. It is characterized frequently by an abrupt invasion, and is marked by rigor, high fever, early nervous symptoms of great prominence, a maculo-petechial eruption appearing between the third and fifth days, and a termination by crisis.

**Historic Note.**—This affection has been known from time immemorial. In 1759 the name *typhus*, which is at present universally employed, was given to it by Sauvages. In presanitary times it prevailed extensively in epidemic and endemic forms, particularly in Ireland and Russia, and also, though less frequently, in the seaport towns of our own country. It constituted one of the chief plagues of the olden times, if not the chiefest, and its devastations among the armies were more destructive of human life than even war itself.

In 1812 typhus fever first appeared in America in the New England States. Its ravages did not cease until every Eastern State had been visited by the plague, when it totally disappeared. In 1836 it reappeared in Philadelphia in virulent form and with deadly effect. It was at this period that Gerhard began his careful studies, which resulted in the separation of typhus from typhoid. During the last half century comparatively few instances of typhus have been met with in this country, though it still appears constantly in certain quarters, abroad (Great Britain, the eastern portion of Germany, Poland, Russia, and some parts of Southern Europe). All isolated cases and small groups of cases that have been observed in very recent times here have been properly attributed to importations from other countries, and chiefly from Ireland. Since the epidemic in 1836 the disease has not gained a foothold on our shores, although in the early part of 1893 it appeared in New York City, and 150 cases resulted.

**Pathology.**—The various viscera present no characteristic lesions. After death the eruption continues to be visible, and often large ecchymoses are observable on the dependent parts of the body.

Certain organs may present pathologic appearances, but they are not constant and are the result of the secondary infection which the typhus invites. The serous membranes—the pericardium in particular, and at times the gastro-intestinal mucosa—are the seat of ecchymoses. There is hyperplasia of the lymph-follicles, but no subsequent ulceration. Hemorrhagic extravasation may also occur into the muscles, the latter being dark and often showing hyaline and granular changes; the *heart-muscle* is especially apt to undergo a granular degeneration. The *spleen* is considerably enlarged, soft (even diffuent at times), and of a dark (frequently bluish) red color. The *liver* is somewhat swollen and may be softened, while the *kidneys* not rarely manifest the changes belonging to nephritis. In other instances they are merely congested. In the *lungs* are found a variety of lesions peculiar to different complicating conditions (bronchitis, lobular pneumonia, lobar pneumonia, pulmonary congestion with or without edema), and occasionally *pleurisy* (sero-fibrinous or purulent) may be present. *Nervous lesions* are conspicuous by their absence. An effusion, either serous or sero-hemor-

rhagic, into the subarachnoid space and the ventricles may be noted, and quite commonly there is *cerebral congestion*. In rare instances there may be a *meningitis*. The *blood-changes* are marked, the color being dark, the fluidity much increased, while the coagulability is greatly diminished; and the intima of the aorta is frequently blood-stained.

**Etiology.**—The direct cause or special micro-organism connected with the typhus contagion has not, as yet, been isolated, notwithstanding the fact that the morphologic and biologic studies of the blood obtained by Brannan and Cheesman from the finger-tips of six patients during the mild epidemic of typhus in 1893 showed the presence of a bacillus that proved pathogenic for rabbits, guinea-pigs, and white mice.<sup>1</sup> Lewaschew<sup>2</sup> has also detected in the blood of typhus patients a distinctive micro-organism. Further observations, however, with a view to showing the constant presence of these micro-organisms in typhus fever, are necessary to demonstrate that they are the specific cause of the disease.

It is a known fact, nevertheless, that when typhus arises in a locality in which it was previously unknown, it is dependent upon a transference of the typhus virus from without, and does not arise spontaneously; this cannot be too strongly emphasized. The different *modes of conveyance of this poison* from one place to another are not known positively, but we can be confident that its source is in a preceding case, and that it may leave the body in the expired air, in the epithelial scales thrown off, and in other excretory or secretory products of the body. The poison is apt to be transmitted by *contagion* from the patient to others who approach him; and there is convincing proof that it may be transferred by means of *fomites* (wearing apparel, articles of furniture, etc.). What its precise *gateway* into the body is we do not definitely know, except that it is more likely to enter through the respiratory tract (by inhalation) than through the alimentary canal.

**Predisposing Causes.**—The influence of *insanitary surroundings* upon the spread of this affection is positive and vital. Among special conditions may be mentioned filth, poverty, famine, and overcrowding, and here it may be inferred that typhus is a disease of the lower classes. Broadly speaking, any condition of the system in which the natural vitality and resistance to bacterial invasion are lowered increases susceptibility to the disease, and among additional influences which possess considerable etiologic influence are overwork, intemperance, depressing emotions, etc.

*Age* has no direct influence. Obviously, however, the young and middle-aged furnish a preponderant proportion of cases, owing to the fact that they are more liable to exposure to the virus than during other periods of life. *Sex* has no positive influence, and the *season* plays only a minor part. Epidemics may, however, occur rather more often in winter than in the other seasons, since the homes of the pauper population are not so well ventilated, and hence are less cleanly in winter than during the rest of the year.

**Clinical History.**—Incubation.—This lasts from nine to twelve

<sup>1</sup> *Annual of the Universal Medical Sciences*, 1893, p. 60, section H.

<sup>2</sup> *Ibid.*, p. 61, section H.



days. There may be prodromal symptoms during the concluding days (one, two, or more of this period), such as anorexia, general malaise, etc., but in most instances *invasion* is sudden.

**Pre-eruptive Stage.**—The early symptoms are either a series of chills or one severe rigor, accompanied by vertigo, tinnitus, headache, muscular pains, profound prostration, and fever. The temperature quickly ascends to a high level, reaching 104° or 105° F. (40° or 40.5° C.) as early as the second or third day. The fever is continuous in type, and in severe cases a serious systemic condition may often be developed. The pulse is accelerated proportionately to the temperature and is of good volume. Bronchitis may be present, the appetite is lost, and the thirst is excessive, while a thick, yellowish-white coating covers the tongue. Vomiting occurs, and may be a prominent symptom. The urine is often scanty, its specific gravity is increased, and it may contain a trace of albumin. The cheeks are flushed and the eyes are injected.

*Nervous symptoms* appear early—in the worst cases at the very onset—and are quite pronounced. At first there may be either mild or active delirium, but soon there is stupor or even actual coma, and the face takes on a dull, stupid look. With few exceptions the spleen on palpation is found to be enlarged.

**Eruptive Stage.**—Between the third and fifth days of the invasion the **characteristic eruption** appears *without an accompanying decline in the temperature*. The rash comes out first upon the trunk, chest, and abdomen, extending thence over the rest of the skin-surface of the body, but, strangely enough, often sparing the face. The crimson-red maculæ are changed in two or three days to a darker hue, becoming hemorrhagic (petechiæ), and when coalescence occurs we have the spotted effect that has caused the name of *spotted fever* to be given to it. This name is also given to cerebro-spinal meningitis, in which the eruption, though it resembles that in typhus fever, does not appear at any given time and is extremely inconstant. Not all of the maculæ are converted, but some may remain as rose-spots, and these disappear when pressed upon, while the petechiæ do not. It is chiefly in the milder grades of typhus that the rose-spots fail to become petechial (*vide infra*). The skin-surface between the spots is sometimes diffusely hyperemic, and the eruption is usually rather abundant, though in well-authenticated cases it has been scanty or even wholly missing. Unlike many other eruptive diseases in the stage of eruption, the symptoms of typhus fever assume an aggravated type in typical and severe cases. The temperature continues high, often reaching 106° F. (41.1° C.) or even higher, with slight nocturnal remissions. The pulse becomes quite rapid (120–140 or more), feeble, and possibly irregular (often dicrotic), and the respirations increase markedly in frequency. At this time severe bronchitis, leading to *broncho-pneumonia*, is apt to occur as a complication. The tongue is brown, fissured, tremulous, and occasionally black and rolled up, without power to protrude from the mouth. Sordes form on the teeth and lips. The urine is scanty, high-colored, and often albuminous, and there may be retention from paralysis of the bladder.

The **nervous disturbance** is intense, and may take the form of typhomania, leading to complete coma or maniacal delirium. The patient

often lies with eyes open, staring into space, yet unconscious and in the condition known as *coma-vigil*. The motor nerves show derangement (tremors, subsultus tendinum, etc.), and carphologia (picking at the bed-clothes) is a common symptom. The decubitus is dorsal, as a rule; the flushed cheeks gradually become dusky, the face expressionless, and the pupils often contracted. The prostration reaches an extreme degree, and absolute exhaustion often terminates life.

As a rule, in favorable cases the end of the febrile period comes by crisis between the fourteenth and seventeenth days of the disease, and the temperature drops in the course of twenty-four or thirty-six hours to normal. Immediately preceding the crisis there is generally a great and sudden rise of the temperature (*perturbatio critica*), and the decline may be interrupted by slight irregularities or fresh exacerbations. The occurrence of the crisis is marked by rapid improvement in the symptoms in general. The stupor suddenly gives place to a clear mind (sometimes following a profound sleep), the eruption fades quickly, the facial phenomena disappear in inverse order of their appearance, and the general strength is rapidly recovered.

**Leading Symptoms and Complications.—Course of the Fever.**—Although the temperature, as stated above, rises rapidly on the first day of the illness, it should be added that the highest grade is usually reached as late as the fifth or sixth day. Maximum temperatures of 105°, 106°, or even 107° F. (40.5°–41.6° C.) are common. Hyperpyrexia usually heralds a fatal termination, the temperature mounting to 108°, 109° F. (42.7° C.), or higher, though in light cases the acme may not exceed 103° F. (39.4° C.). During the height of the affection the temperature pursues the continued type (slight morning remissions), with moderate oscillations, till the occurrence of the crisis which has been described. The fall of temperature may occasionally be more gradual than before indicated, though this is a comparatively rare phenomenon.

The **lungs** frequently present complications (*vide* Pathology), among which the most common are bronchitis, broncho-pneumonia, and hypostatic congestion. Broncho-pneumonia is especially dangerous, its development often preceding a fatal termination, and it may lead to pulmonary gangrene. If the gangrenous, consolidated areas connect with the pleura, empyema commonly results. Sero-fibrinous pleurisy also may occur as a secondary event, as may lobar pneumonia, and to recognize the latter the local physical signs must be fully appreciated, since the rational symptoms are feebly expressed.

The **heart** in typhus continues to grow progressively weaker until, in many cases, a fatal issue is reached. This is manifested by the change in the character of the first sound, which becomes more and more indistinct as the case progresses. A systolic murmur (probably of hemic origin) may be audible at the apex.

The **nervous phenomena** have been sufficiently detailed. *Meningitis* has been met with, but is very rare as a complication. Reference has been made to the occurrence of the ordinary febrile albuminuria in this disease, and it remains to be pointed out that *hemorrhagic nephritis* very rarely intervenes. During the febrile period the uric acid and urea increase in quantity, while the chlorids decrease.



The **digestive tract** rarely presents distressing symptoms and complications. *Hematemesis* is most common, and *cancrum oris* has been noted occasionally. Cases in which the mouth does not receive proper care are apt to develop *parotitis*, which often passes on to suppuration, and *septic processes*, causing abscesses in different parts of the body (joints, subcutaneous tissue, etc.), may arise as complicating events.

Among the *sequelæ*, *neuritis*, followed by *paralyses*, deserves first place, and **gangrene of the remote extremities** (toes, fingers, etc.) has also been observed.

The **general course and duration** of typhus are variable. There is a *mild type* whose course is run in from seven to ten days, and in such the crisis occurs soon after the appearance of the eruption, which may not proceed to the petechial stage. In this type the development of serious symptoms or grave complications is the exception. A *malignant type*, however, also occurs (*typhus siderans*), and this often proves fatal before the time for the appearance of the rash.

Some epidemics are characterized by the relative frequency of light forms, and others by the severer types of the disease.

**Diagnosis.**—On the known presence of an epidemic with special causative factors (unhygienic surroundings, exposure to the poison, etc.), and with the course and characteristic symptoms, the diagnosis of typhus fever can be made. Of special value is the eruption—its time of appearance (third to fifth day), mode of distribution, petechial character, and peculiar behavior under pressure. The recognition of lighter types, on the one hand, and malignant, on the other, is not possible from the symptoms alone, but it is so from the light afforded by a definite knowledge of the existence of an epidemic in the vicinity.

**Differential Diagnosis.**—**Typhoid fever** is distinguished from this affection by (*a*) its gradual onset, unaccompanied by severe rigor; (*b*) the relatively diminished violence and the later development of the nervous symptoms; (*c*) the less intense lumbo-muscular pains; (*d*) the less abundant eruption, which is non-petechial and appears on the seventh or eighth day; and (*e*) the gradual convalescence.

**Cerebro-spinal meningitis** may be distinguished by a more intense headache, by retraction of the head, hyperesthesia, intolerance of sounds, photophobia, palsies of the eye-muscles (*strabismus*), a greater tendency to convulsions, and, finally, by both the absence of the typhus eruption and the countenance absolutely devoid of expression.

**Uremia** is excluded by the absence of the previous history which it always gives (headache, vomiting, and diarrhea extending over a variable period of time), by the presence in typhus of high temperature and a petechial eruption, and by the absence of edema of the extremities and face. Characteristic urinary phenomena are associated in uremia, and it must not be forgotten that among the rarer complications of typhus is *acute hemorrhagic nephritis*.

In **pneumonia** the mode of onset is not unlike that of typhus, but the early development of the local physical signs, the absence of the typhus eruption, and the non-epidemic appearance of the disease are points which serve to distinguish the former from the latter disease.

*Relapses* are among the rarest of clinical events, and one attack, as a rule, bestows immunity for life.

**Prognosis.**—To arrive at a correct prognosis it is necessary to consider (1) the degree of severity of the particular type from which the patient is suffering, (2) the number and character of the complicating conditions present, or likely to occur if the case be of a severe grade, and (3) any peculiar circumstances connected with the individual, among which his food-supply and his sanitary surroundings are deserving of chief mention. In general terms, typhus fever is a grave disease, but its frequency of occurrence, and also its virulence, have been markedly reduced in consequence of better sanitation. The mortality-rate has been, during the last half century, lowered immensely, and is between 10 and 20 per cent. at the present day.

**Treatment.**—This need not be discussed at length, since it embraces, in the main, the same principles that were evolved in the treatment of typhoid fever.

**Prophylaxis** demands *thorough disinfection* and *absolute isolation*. A special hospital for contagious diseases is always to be preferred to the best accommodations obtainable in private families. When, however, patients cannot be transferred to special hospital wards and must be treated in private houses, the sick-room must be kept clean, well-ventilated, and at a temperature ranging from 60° to 65° F. (15.5° to 18.3° C.). No one other than the doctor and nurse should be allowed to occupy or even enter the room. The thorough disinfection already described under Typhoid Fever must be enforced with equal care, and the importance of supplying fresh air to typhus patients has been abundantly shown by the great reduction in the mortality-rate among those treated in tents as compared with that in the hospital wards.

The general management, including the use of stimulants, in this disease does not differ from that advised in typhoid fever, except that a more prompt return to solid food can be made during convalescence than in typhoid. Fresh water should be given freely, and, in view of the blunted sensibilities of the patient, should be offered at regular intervals. *Hydrotherapy* constitutes the best means at our command for controlling (by virtue of its stimulating effect upon the cardiac and respiratory centers) the temperature and the nervous symptoms, while at the same time it obviates dangerous complications. In addition, the use of antiseptic agents and tonic measures is to be recommended. The fact that typhus is a self-limiting affection, and therefore curable if life can be spared until it has run its usual course, gives those measures that are intended to combat exhaustion high rank in the treatment of this affection.

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## RELAPSING FEVER.

(*Febris Recurrens*; *Relapsing Typhus*.)

**Definition.**—An acute infectious disease caused by the spirillum of Obermeier, and characterized by febrile periods which usually last six days, and are separated by afebrile periods of the same duration.

**Historic Note.**—The first accurate account of this affection was published in 1739, though it is known to have prevailed in Europe and



Ireland prior to that period. During the next century numerous epidemic outbreaks, more or less extensive, occurred, and in 1844 the disease made its first appearance in America at the Philadelphia Hospital, being brought by immigrants from Ireland. Subsequently small groups of cases occurred, and were reported by Flint and others, and in 1869 it prevailed considerably in Philadelphia (where it was studied especially by E. Rhoads and William Pepper) and in other large cities of the country. This was the last epidemic appearance of the disease in the United States, though in the years 1885 and 1886 Russia was visited by an epidemic of considerable magnitude.

**Pathology.**—The solid organs of the body present no characteristic anatomic changes, though when death occurs during the febrile period the various viscera (heart, liver, kidneys) are the seat of cloudy swelling, and sometimes of hemorrhagic infarct and extravasation. The *spleen* shows the most constant alterations, being enlarged, but in size it exhibits a great variability. Infarction is frequent, and the lymphoid element of the bone-marrow often shows hyperplasia. If jaundice has been present during life, it is visible after death.

**Etiology.**—**Bacteriology.**—In 1873, Obermeier discovered in the blood of patients suffering from relapsing fever a special organism, the *spirillum Obermeieri*, and subsequent investigations by others have fully confirmed his observations with reference to the causal relation of this micro-organism to relapsing fever. The specific agent, or *spirocheta*, is a delicate filamentous organism of spiral form and much elongated, its length equalling four to six times the diameter of a red blood-corpuscle (Fig. 8). Examined under the microscope during a pyretic period, it is seen to exhibit active motion among the blood-cells, this motion being spiral and following the long axis of the organism. It is *aërobie*, and may be demonstrated in *dry blood* by staining with anilin colors, but the spirillum has never been found in other fluids or secretions of the body. It is also apparent in the blood only during the paroxysms, and Dr. Van Dyke Carter's careful studies have shown that by inoculation of the blood containing spirillar organisms or their germs the disease may be conveyed to new or old subjects. Shortly before the crisis the spirilla disappear from the blood, and are, as a rule, absent during the whole of the succeeding apyrexial period, and inoculation now fails to produce the disease. After death they are found in all the organs, but they have not been cultivated successfully on artificial media, and little is known of their life-history.

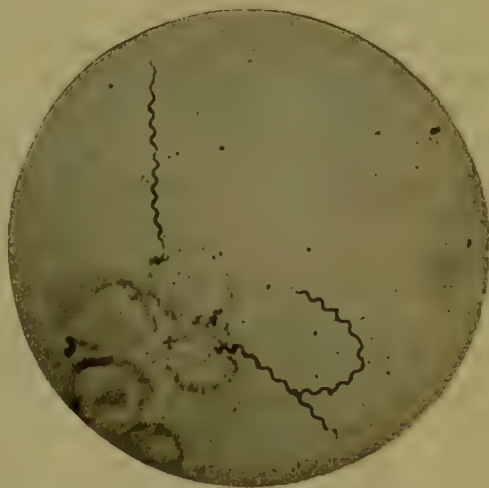


FIG. 8.—Bacillus of relapsing fever (from human blood);  $\times 1000$  (Günther).

**Predisposing Causes.**—**Age.**—The complaint is most common in young adults between fifteen and twenty-five years.

**Sex.**—A larger proportion of males than females is affected.

The disease is especially apt to prevail in times of famine, and, in short, the same unhygienic surroundings that produce typhus also predispose to relapsing fever.

**Clinical History.**—The **incubation** period ranges in its duration from four to ten days, though sometimes it is even briefer; and in this stage certain symptoms (malaise, fugitive pains, etc.) may appear.

The **invasion** is quite abrupt, often occurring on awakening in the morning, and commonly the attack is ushered in with a severe rigor, though there may be only a repeated slight shivering. The chief accompanying symptoms are frontal headache, vertigo, severe pains in the loins and limbs, and marked physical prostration. The temperature rises soon, and often rapidly, reaching  $105^{\circ}$ – $106^{\circ}$  F. ( $41.1^{\circ}$  C.), or higher still, on the first or second day. The skin is dry and pungent, and presents very soon either a “characteristic dirty-yellow color” or a distinctly bronzed appearance. The cheeks are flushed, the eyes sunken, and profuse perspirations often take place (sometimes alternating with chills), in consequence of which sudamina are frequently observed. Other forms of eruption have been described, but none that are either constant or characteristic. In certain epidemics *herpes labialis* has been very generally noticed. At first the *tongue* is moist and coated with a yellowish-white fur, and later it may become brown, dry, and fissured, with sordes on the teeth. *Ulcerative stomatitis* has been observed occasionally, and catarrhal pharyngitis and mild tonsillitis may be evidenced by pain on swallowing and other symptoms. Among the earlier symptoms are excessive thirst, anorexia, nausea, and vomiting. The vomitus may be yellowish-green, green, or even black in color, and consist of bile in varying proportions (rarely, also, blood) and gastric secretions. Constipation often precedes invasion, and is apt to continue throughout the attack.

The pulse rises rapidly with the temperature, though the normal ratio between the two is not maintained. At first the pulse is full and strong, and its beats number from 100 to 140 or more per minute; but in serious cases it becomes weak, irregular, or even intermittent, while at the same time the heart-sounds grow more and more feeble and indistinct. Hemic murmurs may be audible. The *nervous* derangements are not of a grave character, but the headache persists and is severe throughout, and the patient is restive and sleepless. Delirium is not common, and, though occasionally this symptom assumes a prominence toward the crisis, the intellect remains clear as a rule. The urine presents the ordinary febrile characteristics, and may contain albumin and casts. It also contains bile-pigment when jaundice is present. The respirations are accelerated, and immediately preceding the crisis urgent dyspnea may be developed.

The **physical signs** during the febrile paroxysms are few. The epigastric region and the nerve-trunks are tender to the touch, while the skin-surface and certain muscles are often hyperesthetic. *Palpation* detects a variable degree of enlargement of the spleen and liver, and the signs of bronchitis, of lobular pneumonia, and of hypostatic congestion of the lungs may be present. The symptoms above detailed persist with slight daily fluctuations of temperature till there occurs a turning-point.



**The Crisis.**—This occurs from the fifth to the seventh day, and rarely as late as the tenth. It is sometimes heralded by a critical rise of temperature, the mercury touching  $108^{\circ}$  F. ( $42.2^{\circ}$  C.), but evidenced chiefly by a rapid fall of temperature (within twelve hours) to or below the normal, with profuse sweating. Coincidentally, all other symptoms disappear with marvellous rapidity. The critical sweat may be replaced by diarrhea, intestinal hemorrhage, metrorrhagia, or epistaxis, and then follows a speedy afebrile convalescence, so that after the lapse of a day or two the patient expresses himself as being well.

During the intervals between the paroxysms the skin may exhibit a faintly jaundiced tint; there may be trivial evening exacerbations of temperature, particularly if complications be present and outlast the fever stage; and the spleen is evidently enlarged. There may be, though rarely, but a single paroxysm. As a rule, at the expiration of the second week there will be a recurrence of all the active symptoms of the primary attack, including the rigor or fits of chilliness and fever. Quite frequently a third pyrexial stage takes place, and rarely a fourth or even fifth.

The *duration* of the first relapse is briefer than the primary pyretic stage, and if there be subsequent relapses, each succeeding one is separated from its predecessor by the usual apyrexial period, but is briefer

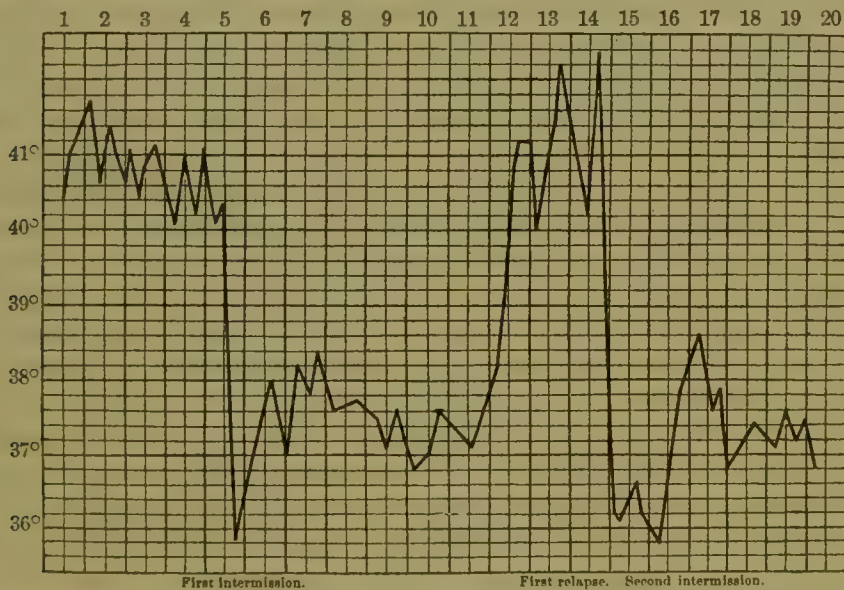


FIG. 9.—Temperature-curve of relapsing fever.

and lighter. Hence, should a fourth or a fifth febrile period occur, it is, as a rule, quite rudimentary. The relative duration and severity of the different febrile periods, their manner of recurrence, and the course of the fever are considerations that can best be appreciated by a glance at the accompanying temperature-chart (*vide* Fig. 9).

**Complications.**—These are not frequent. At the head of the list stands lobar pneumonia, and next comes broncho-pneumonia, which is always secondary. Other conditions, belonging to the latter class, are septico-pyemic processes, iritis, irido-choroiditis, suppurative parotitis, laryngitis, entero-colitis, and neuritis. In pregnant women abortion

may take place. Epistaxis has been noted, and has even proved dangerous in some epidemics. Acute hemorrhagic nephritis is a very rare but serious complication when it does occur, and may be dependent upon a primary affection. As the result, most probably, of the very high temperature the heart may become exhausted, and the occurrence of sudden paralysis is not unknown.

**Clinical Varieties.**—The difference in the general course of cases in different epidemics, and even in the same one, is, for the most part, the direct result of the varying degrees of intensity of the infection. Thus very light or even rudimentary cases occur in which the whole course may be made up of one or two brief febrile periods, and their resemblance to ordinary intermittents may be close. The so-called "*bilious typhoid*," which is a form of relapsing fever, occupies the other extremity, being of malignant type. It is sometimes characterized by the usual symptoms of the disease, only greatly intensified; but more often, perhaps, the condition early merges into a typhoid state, to which are added certain grave features and complications (marked icterus, hematemesis and hemorrhages from other outlets of the body, uremia, sudden collapse, etc.). Septic and pyemic processes, including infarctions, are common accompaniments, and the outcome is frequently unfavorable.

**Diagnosis.**—The prevalence of an epidemic in which the cases present similar symptoms; the sudden onset; the course and intensity of the fever with its concomitants; the termination by crisis on or about the seventh day; and the peculiar manner of repetition of the fever-attacks after an afebrile period of equal duration,—are points that distinguish relapsing fever from other affections which simulate it more or less closely. Additional symptoms that are of special value for diagnosis are—enlargement of the spleen and liver, a negative character of the nervous and a prominence of the gastric phenomena, and jaundice. To be able to state that relapsing fever is positively present the *spirocheta Obermeieri* must be found in the blood, and this is particularly true in the earlier cases of an epidemic, before they have passed through their typical relapses. To demonstrate the presence of this parasite in the blood during the fever-stage is not a difficult task. A drop of blood obtained from the finger-tip is to be examined microscopically without previous dilution. On account of their size and motility the spirilla can be readily detected, and usually the attention of the examiner is first arrested by the peculiar joggling movements of the red blood-corpuscles. Then the real disturbing agents appear as slender spirals with a snake-like motion. Their identity may be confirmed by staining with anilin colors, and, in exceptional cases, by injecting them into the blood of the monkey, in whom they produce the disease.

**Differential Diagnosis.**—*Typhus fever* may be mistaken for relapsing fever, since both have the same predisposing causes, both prevail epidemically, both are characterized by an abrupt onset, with or without prodromes, and by a continued type of fever. On the other hand, certain points of distinction serve to separate them reliably. In relapsing fever the eyes are clear but hollowed, the cheeks are flushed, and there is a dirty-yellow tint of skin; in typhus the eyes are injected, the pupils contracted, the face wears a stupid, inanimate expression, and there is in addition the characteristic maculo-petechial eruption. In



relapsing fever the intellect remains clear, or there may be delirium toward the height of the febrile paroxysm; in typhus stupor develops early, and later there is coma or coma-vigil, with acute or low-muttering delirium and adynamic symptoms. In the former disease the primary period of fever is briefer than in typhus by a ratio of 7 to 14. Relapses are the rule in relapsing fever, and the exception in typhus; while in the blood of relapsing fever patients may be found the spirillum, which is absent in typhus fever.

Pel and Ebstein have described a febrile condition which sometimes occurs in pseudo-leukemia and simulates that of relapsing fever; but it may be distinguished by the absence of the spirilla from the blood, the general enlargement of the lymphatic glands, as well as of the liver and spleen, and the fact that the pyrexial periods do not tend to grow shorter.

**Prognosis.**—The prognosis of relapsing fever is good, but of “bilious typhoid” it is bad indeed. Apart from the type, we must consider, in this as in all other acute infectious diseases, the number, character, and frequency of occurrence of the various complications. As stated, these are few, infrequent, and mostly benign. Among those signaling danger are severe hemorrhages (epistaxis, metrorrhagia, hematemesis, etc.), premature labor, signs of uremia and syncope, marked jaundice and excessive vomiting, urgent diarrhea, etc. Perhaps the most frequent causes of death are pneumonia and acute hemorrhagic nephritis. Individual circumstances exert an influence upon the prognosis, and of those that render it more grave are the want of good nursing, privation, a previously enfeebled system, and old age (the disease being more fatal in elderly than in younger subjects).

The duration depends upon the number of paroxysms, since the latter are of definite length. In the majority of cases there is but one relapse, and in this event the disease lasts from eighteen to twenty days.

**Treatment.**—The general management, including the time and use of stimulants, must be based on the same principles as are employed in typhoid fever. The fever, as well as the nervous and other leading symptoms, is to be opposed by the cold or gradually cooled bath, employed as indicated in the article on the treatment of the latter disease. If, as may happen, there are adequate reasons why balneo-therapeutics cannot be used, then cold spongings, with the ice-cap or the cold pack, may be tried. Internal antipyretics may be reserved for use in cases in which the temperature is very high and the above-mentioned means are impracticable. Small doses of phenacetin (gr. ij to v—0.1296 to 0.3240) or acetanilid (gr. ij to iij—0.1296 to 0.1944) are to be administered, at the same time guarding the heart, and the signs of collapse must be promptly met by the free yet prudent use of stimulants (strychnin, alcoholics, ammonium, etc.). Vomiting often induces marked debility, and calls for the use of ice or iced champagne and small doses of cocain, morphin, or dilute hydrocyanic acid, preceded by a mercurial laxative. Counter-irritation over the epigastrium is also useful. For the intense muscular pain, restlessness, and sleeplessness nothing is so good as morphin given subcutaneously, and Dover's powder may be employed if the pain be of moderate severity. During the intermissions the patient should be kept indoors for ten days or more, lest exposure or sudden exertion

predispose him to a relapse. Solid food may now be gradually resumed, and tonics judiciously given. The treatment of *relapses* differs in no way from that of the first febrile period.

## MALARIAL FEVER.

(*Chills and Fever; Fever and Ague; Swamp Fever.*)

**Definition.**—An infectious, non-contagious disease caused by the hematozoa of Laveran. It is characterized by splenic enlargement, brief febrile attacks which recur periodically, melanemia, and a tendency in protracted cases to irregular fever and extreme anemia. The following sub-varieties will be discussed: (I.) *Intermittent fever*; (II.) *Pernicious intermittent*; (III.) *Remittent fever*; (IV.) *Malarial cachexia*; (V.) *Masked intermittents*; and (VI.) *Malarial hematuria*.

**Historic Note.**—There are few diseases with which the profession has been acquainted longer than with the more typical forms of malaria, and chief among the earliest known hot-beds of this disease were the city of Rome, the Pontine marshes about the latter, and the swamps along the lower Danube. Except in the extremes of latitude there are few localities in which malaria has not been endemic, with seasonal epidemic outbreaks; yet it is pretty generally believed that the prevalence of the disease long has been, and still is, diminishing. This view is fully corroborated by my own observations.

A similar progressive decrease, with slight annual variations, was noted during a period of five years (from 1885 to 1889) when the cases from four leading hospitals of Philadelphia were considered together. The total number of cases for this space of time was 1132. It was also found that a tracing representing the number of cases of malaria admitted into the Pennsylvania Hospital yearly during the period extending from 1853 to 1893, inclusive, showed a similar tendency to decline, though in a somewhat less striking degree. Osler has called attention to the fact that the diagnosis of malaria was much more frequently made before the discovery of the parasite than has since been the case, and that, therefore, early statistics of this disease are apt to be misleading.

New England, once a region in which the disease was very prevalent, now affords few cases. In the southern portion of the United States, also, the severer forms of malaria prevailed extensively, but a marked tendency to progressive reduction in the number of cases has also been observed here. It must not be forgotten, however, that in some districts of the United States, from which malaria had disappeared, it has reappeared, while other localities, formerly free from the disease, have become more or less malarious. In foreign lands (England, France, Germany, etc.) the constantly decreasing prevalence and virulence of this disease have been noted by numerous careful observers.

**Pathology.**—The chief and most constant morbid lesions are attributable to the direct effect of the malarial parasites upon the blood.



The symptomatic anemia (often quite pronounced) results from the destruction of red corpuscles, which may be observed in all stages, by the parasites. There is a marked tendency to an accumulation of pigment in the blood and in certain of the internal organs, particularly the spleen and liver. To account for this is the fact mentioned in the description of the amebæ (*infra*) that the hemoglobin of the blood is converted into melanin (pigment) by the organisms. The malarial parasite also engenders a toxin which may be in part responsible for the morbid lesions of the disease.

*The spleen* is engorged with blood, and at first is swollen (chiefly during the febrile paroxysm), but it soon becomes permanently enlarged ("ague-cake"). A rare accident in intermittent fever is rupture of the spleen. Hemorrhagic infarcts are occasionally presented by this organ.

*The liver* is also engorged, but not to the same extent as the spleen.

*The heart-chambers* may be found to be acutely dilated.

**Etiology.**—(1) **Soil.**—It is a generally accepted fact that a certain condition of the soil, especially as regards a considerable humidity, is essential to the development of the malarial poison. Marshes are often highly malarious, and particularly the swamps, that are overflowed at certain seasons and exposed to the influence of the atmosphere at certain other seasons, are breeding-places for malaria. Fresh-water marshes favor the development of the malarial organism, and are most fruitful in influencing its growth when located near the coast and tainted with salt water. Again, marshy districts affording luxuriant vegetation are notorious as malarial foci. Keeping in remembrance the foregoing facts, we can readily see why malaria is unusually prevalent in certain countries (chiefly tropical), and why it is confined to the low-lying estuaries and the deltas of rivers. The same facts explain satisfactorily why certain districts which were very liable to the affection should have become, as the result of denudation of the virgin soil and its subsequent drainage and cultivation for longer or shorter periods, entirely free from the complaint. On the other hand, the upturning or removing the surface of the virgin soil may be followed by the appearance of malaria in localities in which it has been previously unknown. While we regard the soil as the natural "home and cradle" of the malarial poison, we are not acquainted, as yet, with all of the telluric conditions upon which its presence or absence depends; and it is an error even to regard all marshy districts as being necessarily malarial, since the disease has been met with on dry, sandy soils, and even on distinctly rocky strata.

(2) **Climate.**—Malaria is more prevalent in tropical and subtropical than in temperate climates, and more common in the latter than in the polar zones. Hence it occurs more frequently in the southern than in the northern States of our own country. *Temperature, per se*, constitutes the indispensable factor, the virus of malaria being inactive at temperatures below 65° F. (18.3° C.), while very high temperatures may arrest the *plasmoidal growth*. For the *development and propagation* of the malarial parasite the presence of an abundance of atmospheric oxygen is also apparently essential, though the influence of *moisture* in the air upon the malarial germ is little understood.

(3) The **winds** may transfer the poison from place to place, but trees planted in rows or clumps often successfully intercept the poison as it

is being dispersed by air-currents through the lower strata of the atmosphere. Strong winds tend to dry the soil by hastening the surface evaporation, and become an influential factor in combating one of the conditions of soil essential to the development of malaria.

(4) **Rapidly-growing trees** also dry the soil by absorbing enormous quantities of water. They are probably efficient, however, only in localities that have no natural subsoil drainage<sup>1</sup>—a condition often met with in malarial districts. In the Roman Campagna extensive experiments have been made with the eucalyptus tree, and the results have been remarkable, districts protected in this manner becoming almost entirely free from malaria in a few years.

(5) **Seasons.**—In temperate latitudes most cases are developed in the autumn, the maximal period corresponding with the month of Septem-

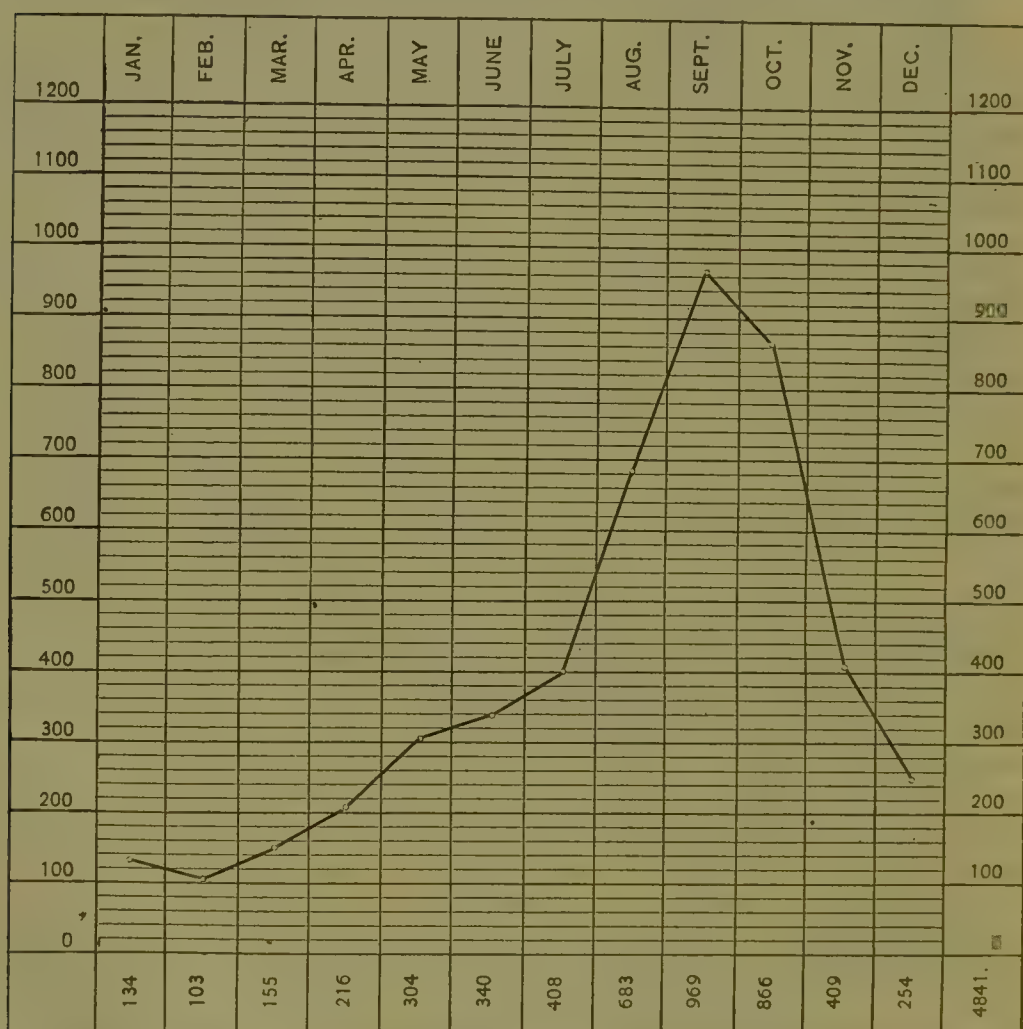


FIG. 10.—Chart showing the seasonal variations of malarial fever. The line increases in increments of 100.

ber, as is shown by the tracing on the accompanying chart (Fig. 10), which is based upon 4841 cases of malaria gathered from the records of the leading Philadelphia hospitals.

Authors who state that malaria is more prevalent in the spring and

<sup>1</sup> *House-plants as Sanitary Agents*, by the Author, p. 263.





## DESCRIPTION OF PLATES I. AND II.<sup>1</sup>

The drawings were made with the assistance of the camera lucida from specimens of fresh blood. A Winckel microscope, objective  $\frac{1}{4}$  (oil immersion), ocular 4, was used. Figures 4, 13, 23, 24, and 42 of Plate I. were drawn from fresh blood, without the camera lucida.

### PLATE I.

#### THE PARASITE OF TERTIAN FEVER.

- 1.—Normal red corpuscle.
- 2, 3, 4.—Young hyaline forms. In 4, a corpuscle contains three distinct parasites.
- 5, 21.—Beginning of pigmentation. The parasite was observed to form a true ring by the confluence of two pseudopodia. During observation the body burst from the corpuscle, which became decolorized and disappeared from view. The parasite became, almost immediately, deformed and motionless, as shown in Fig. 21.
- 6, 7, 8.—Partly developed pigmented forms.
- 9.—Full-grown body.
- 10-14.—Segmenting bodies.
- 15.—Form simulating a segmenting body. The significance of these forms, several of which have been observed, was not clear to Drs. Thayer and Hewetson, who had never met with similar bodies in stained specimens so as to be able to study the structure of the individual segments.
- 16, 17.—Precocious segmentation.
- 18, 19, 20.—Large swollen and fragmenting extracellular bodies.
- 22.—Flagellate body.
- 23, 24.—Vacuolization.

#### THE PARASITE OF QUARTAN FEVER.

- 25.—Normal red corpuscle.
- 26.—Young hyaline form.
- 27-34.—Gradual development of the intracorpuseular bodies.
- 35.—Full-grown body. The substance of the red corpuscle is no more visible in the fresh specimen.
- 36-39.—Segmenting bodies.
- 40.—Large swollen extracellular form.
- 41.—Flagellate body.
- 42.—Vacuolization.

### PLATE II.

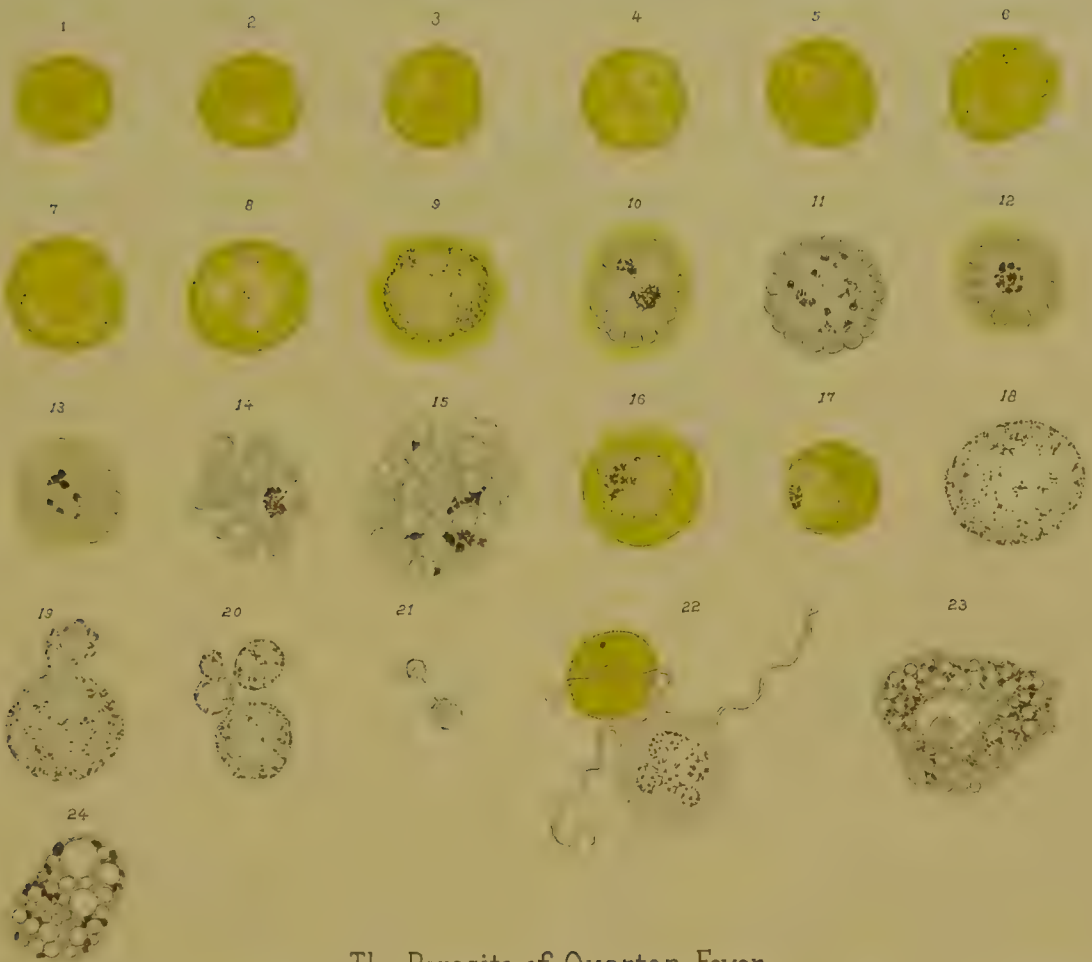
#### THE PARASITE OF ÆSTIVO-AUTUMNAL FEVER.

- 1, 2.—Small refractive ring-like bodies.
- 3-6.—Larger disk-like and ameboid forms.
- 7.—Ring-like body with a few pigment-granules in a brassy, shrunken corpuscle.
- 8, 9, 10, 12.—Similar pigmented bodies.
- 11.—Ameboid body with pigment.
- 13.—Body with a central clump of pigment in a corpuscle, showing a retraction of the hemoglobin-containing substance about the parasite.
- 14-20.—Larger bodies with central pigment clumps or blocks.
- 21-24.—Segmenting bodies from the spleen. Figs. 21-23 represent one body where the entire process of segmentation was observed. The segments, eighteen in number, were accurately counted before separation, as in Fig. 23. The sudden separation of the segments, occurring as though some retaining membrane were ruptured, was observed.
- 25-33.—Crescents and ovoid bodies. Figs. 30 and 31 represent one body, which was seen to extrude slowly, and later to withdraw, two rounded protrusions.
- 34, 35.—Round bodies.
- 36.—"Gemmation," fragmentation.
- 37.—Vacuolization of a crescent.
- 38-40.—Flagellation. The figures represent one organism. The blood was taken from the ear at 4.15 p. m.; at 4.17 the body was as represented in Fig. 38. At 4.27 the flagella appeared; at 4.33 two of the flagella had already broken away from the mother body.
- 41-45.—Phagocytosis. Traced with the camera lucida.

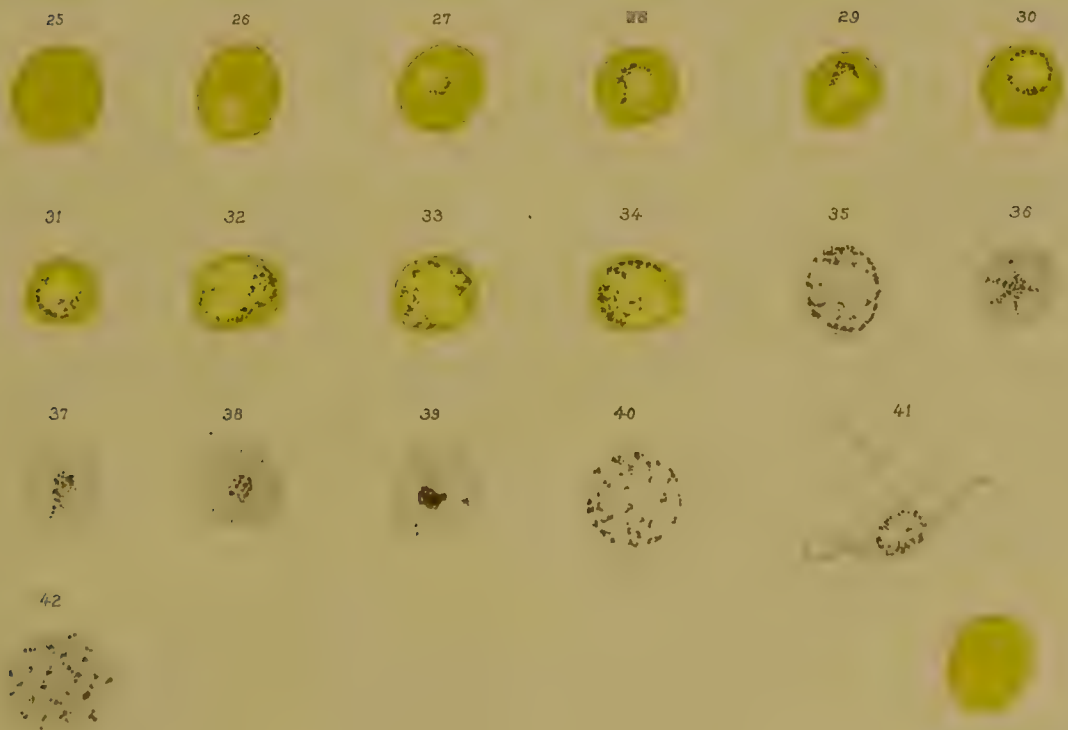
<sup>1</sup>These illustrations are reproduced by permission from the article by Drs. Thayer and Hewetson in *The Johns Hopkins Hospital Reports*, vol. v., 1895.



The Parasite of Tertian Fever.



The Parasite of Quartan Fever.





The Parasite of Aestivo Autumnal Fever.







autumn than in summer and winter in temperate regions are probably in error. An inspection of the tracing will convince the most skeptical that the spring, unlike the autumn, is unattended with increased prevalence of the disease, which is in abeyance not only during the winter, but also practically during the spring, although the cases are seen to increase during the latter period. In the tropics the case seems to be different, and two maximum periods—spring and autumn—and two minimum—summer and winter—obtain. Autumn has, however, the greater number of cases.

(6) **Gravitation.**—The malarial poison escapes from the soil into the superjacent strata of air. That it does not rise far above the earth's surface is shown by the fact that persons occupying the upper stories of a house or living on slight elevations are affected with relative infrequency.

(7) **Race** exerts little influence in other lands than our own, but in the United States negroes are less susceptible than are the whites.

(8) **Sex** is without effect when men and women are equally exposed. Cases are, however, vastly more frequent among males because of their increased liability to exposure, and particularly while following certain occupations (agriculture, marsh-draining, etc.). The 5044 cases collected by myself gave the numerical proportion of 6 to 1 in favor of males.

**The Malarial Parasite.**—In 1879, Klebs and Tommasi Crudeli isolated a low vegetable organism—the bacillus malarix—and claimed it to be the special agent producing all forms of malaria. The evidence afforded by subsequent experiments of other observers, however, failed to corroborate their investigations, and it remained for Laveran in 1880 to discover the specific parasite in the blood of patients affected with malaria. The announcement of his discovery failed to attract widespread attention until 1883, when Marchiafava, Celli, and Golgi published the results of their confirmatory investigations. Since then the claims of Laveran have been abundantly corroborated by Councilman, Osler, James, Dock, Koplik, and others in the United States, by Van Dyke Carter in India, and, more recently, by numerous French, English, German, and Russian observers. It would seem, therefore, as though the evidence as to the specificity of this organism were almost complete, and, at all events, it has invariably been found to be associated with the different forms of malaria.

The malarial parasite belongs to a sub-class of the protozoa known as hematozoa. Of the latter, three varieties, corresponding with the three leading clinical forms of the infection, have been distinguished, and the evolution of two of these parasites at least takes place within the red blood-corpuscles. They enter the red cells in the form of small, non-pigmented plasmodia, exhibiting ameboid motion, and then feed upon their host, transforming, at the same time, the hemoglobin of the latter into dark pigment-granules as they develop. When the intra-globular plasmodia have consumed the red blood-corpuscles the granules of pigment accumulate in the center of the parasite, while on its periphery the processes of subdivision and sporulation are taking place, forming fresh generations of hematozoa. These young parasites assume the form of minute, more or less spheric, hyaline bodies, which again

enter the red blood-corpuscles and start on a new cycle of development. It is probable, as Golgi suggests, that the third variety is not intimately connected with the circulating medium, but that its evolution principally takes place in the internal organs (spleen, bone-marrow, etc.). The special varieties of the malarial parasite will be described separately.

(1) *The Ameba causing Tertian Intermittent Fever.*—This begins its cycle of evolution in the red blood-corpuscle as a small pigmented ameba. Its development is attended with the appearance in its interior of fine, brown, motile granules in the form of pigment, and when matured it about equals the size of a normal red corpuscle. It now assumes a spheric form, the pigment collecting centrally, and sporulation into fifteen to twenty or more segments follows. The tertian parasites are exceedingly numerous in the blood, and pass through the various stages of their life-cycle almost simultaneously, the sporulation of an entire generation occurring within the space of a few hours (Golgi). The occurrence of the malarial paroxysm follows the process of sporulation, which is attended, most probably, with the development of a *toxin*, and the symptoms of the disease may be attributable chiefly to the effects of the latter. The red corpuscle that includes the parasite becomes enlarged and decolorized as the latter develops. The parasite of tertian intermittent runs its cycle in about forty-eight hours. Hence infection by a single generation would result in sporulation every second day, followed by the malarial paroxysm. Quite commonly, infection by two groups of parasites occurs on successive days, and, since each has a definite period of evolution, a daily malarial paroxysm is the result (quotidian intermittent). Multiple infection with this parasite may occur, but with great rarity.

(2) *The Ameba causing Quartan Fever.*—This cannot be distinguished from the tertian parasite at the beginning of its brief career, but later differences are clearly perceptible. Its ameboid movements are more deliberate, and its pigment-granules are coarser, darker, and also less motile than those of the tertian organisms. Unlike the latter, it does not attain the size of the red corpuscles, and during sporulation the segments (five to ten in number) encircle in an orderly way the central pigment-mass or clump, "rosettes" of great beauty thus being formed. The red blood-corpuscle that harbors the quartan parasites contracts upon its destroyer, appears shrivelled, and its color changes at the same time from the normal to a deep greenish or bronzed tint. It sporulates about seventy-two hours after it enters the red corpuscle; hence, if only one group of parasites be present, febrile attacks occur every fourth day, forming the simple quartan intermittent. On the other hand, double quartan infection results in paroxysms on two successive days, followed by an intermission lasting one day, while triple infection, or the presence of three groups, causes daily paroxysms—the quotidian intermittent. Infection by more than three groups of the quartan parasite may occur, but is very rare.

(3) *The Ameba causing Estivo-autumnal Fevers.*—The cycle of this variety is evolved, chiefly, in certain of the internal viscera, and the microscopic examination of the blood in the various stages of the disease does not give a positive result, as in the tertian and quartan types.



The organism invades the red blood-corpuscle, but to what extent is not definitely known. It is a quite small hyaline body, its size at maturity scarcely equalling one-half the dimensions of the red corpuscle, and it accumulates very few fine pigment-granules. The parasite is not to be found in the later stages, except in the blood from certain internal viscera, such as the spleen, bone-marrow, etc. After the condition has lasted a week or more characteristic oval and crescentic bodies, which are more or less refractive, may be observed in the fresh blood. These so-called "sickle-form bodies" show central rods and clumps of coarse pigment, and are especially connected with this category of malarial fevers. The red corpuscle, at whose expense the parasite develops, assumes a brassy hue, often becoming shrivelled and sometimes notched.

The time occupied by the life-cycle of this parasite is still an unsettled question, but it is generally believed to vary between the extremes of twenty-four and forty-eight hours. In one of my own cases the febrile paroxysms recurred every seventy-two hours. For the differences in the period of evolution there is no satisfactory explanation, though the variation may be connected with the circumstance that it frequently (though by accident) penetrates into the red blood-corpuscle.

*Cilia*, or *flagellæ*, exhibiting active motion, may grow from all of the before-mentioned varieties, and not infrequently they become detached and float free in the blood-stream. They are most common in blood aspirated from the spleen, but their true significance is not known. The ciliated forms, according to Manson, probably do not exist in the blood inside the body, but develop very shortly after it is drawn, especially in the estivo-autumnal type. Usually the presence of the cilia is only indicated by occasional oscillatory movements of the red blood-cells surrounding the parasite.

**Mode of Infection.**—The exact manner in which the malarial parasites are transferred from the sick to the healthy is not well understood. It is known to a certainty, however, that the disease can be communicated by injecting the blood of a patient into a healthy individual. On the other hand, it is in no sense a contagious disease, since the poison, after it enters the body of a human being or inferior animal, cannot escape in an active form from the latter into the surrounding atmosphere. Most probably the *poison enters* the system through the *medium of inhalation*, and rarely through the digestive tract.

**Immunity.**—Persons who have had malaria are more liable to fresh attacks than before, although they experience no inconvenience so long as they reside in a non-malarial district.

**Incubation.**—The period of incubation varies in different cases and according to the different clinical types. Thus it is, on the whole, briefer in the remittent than in the intermittent forms of malarial disease, the time usually ranging from five to twenty or more days.

(I.) **Intermittent Fever.**—**Symptoms.**—The clinical history presents itself under two heads: (a) the paroxysms, and (b) the manner in which the paroxysms recur.

(a) *The Paroxysms.*—There may be premonitions lasting from one to several days, and most significant, yet not distinctive, are headache, pain in the nape of the neck, yawning, a yellowish complexion, and a slight splenic enlargement. In a large proportion of the cases, how-

ever, the onset is abrupt. Typical paroxysms present three stages—*chill*, *fever*, and *sweating*. The chill is intense, causing more or less shivering, and often chattering of the teeth. Malaise is marked, the skin is cool and pale, and the face slightly cyanotic. This stage usually occurs in the morning hours, though the time of onset is by no means constant; its duration, also, varies greatly, generally lasting from one to two hours. The internal temperature rises rapidly during the time of the chill.

The *hot stage* succeeds the chill, and, in striking contrast with the first stage, the face wears a decided flush and the skin is burning hot to

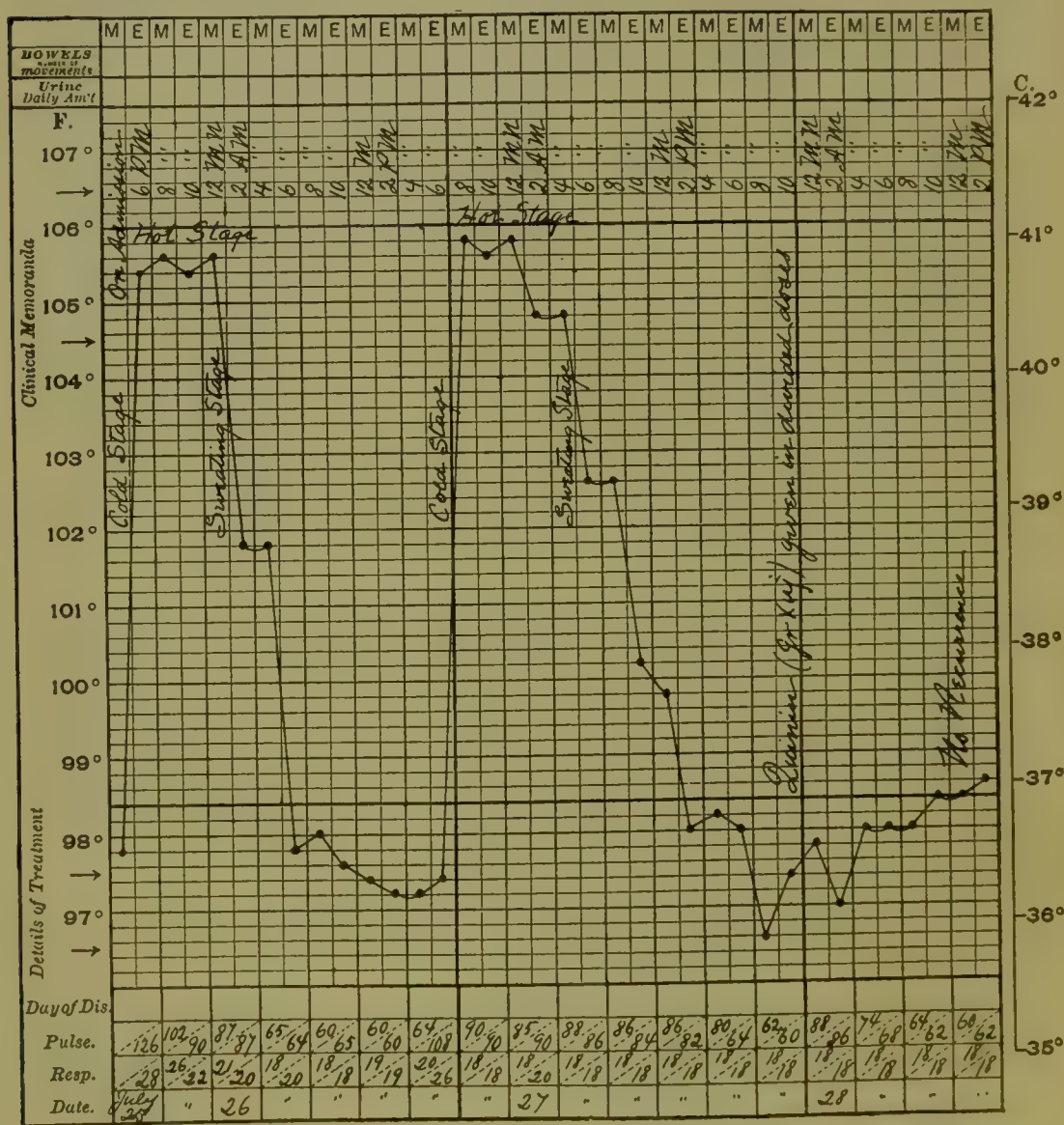


FIG. 11.—Temperature-curve in a case of double tertian fever. C. F. C., aged forty-one years.

the touch. The temperature continues to rise, but not so rapidly as in the first stage. Its maximum level, usually from 104° to 106° F. (40° to 41° C.), is soon reached, and may either be maintained uniformly for several hours, or the curve may show two small summits if the temperature be recorded frequently (Fig. 11). The pulse is full and bounding,



except in the rare instances in which acute dilatation of the heart ensues, when it is quite feeble and sometimes irregular. The length of the second stage exceeds that of the first, being from three to six hours. The temperature generally begins to decline before the close of the febrile stage.

When *sweating*, which soon becomes profuse, sets in, the symptoms of the hot stage are promptly relieved. The temperature falls by crisis, touching the normal level in a few hours; the decline, however, is less rapid than the rise at the beginning of the paroxysm. The fall may be unbroken by any fresh elevations of temperature, though more often the latter occur, and less frequently defervescence occurs by steps, the temperature dropping one or more degrees, and remaining at the new level for a short period (Fig. 12). It again drops about an equal distance,

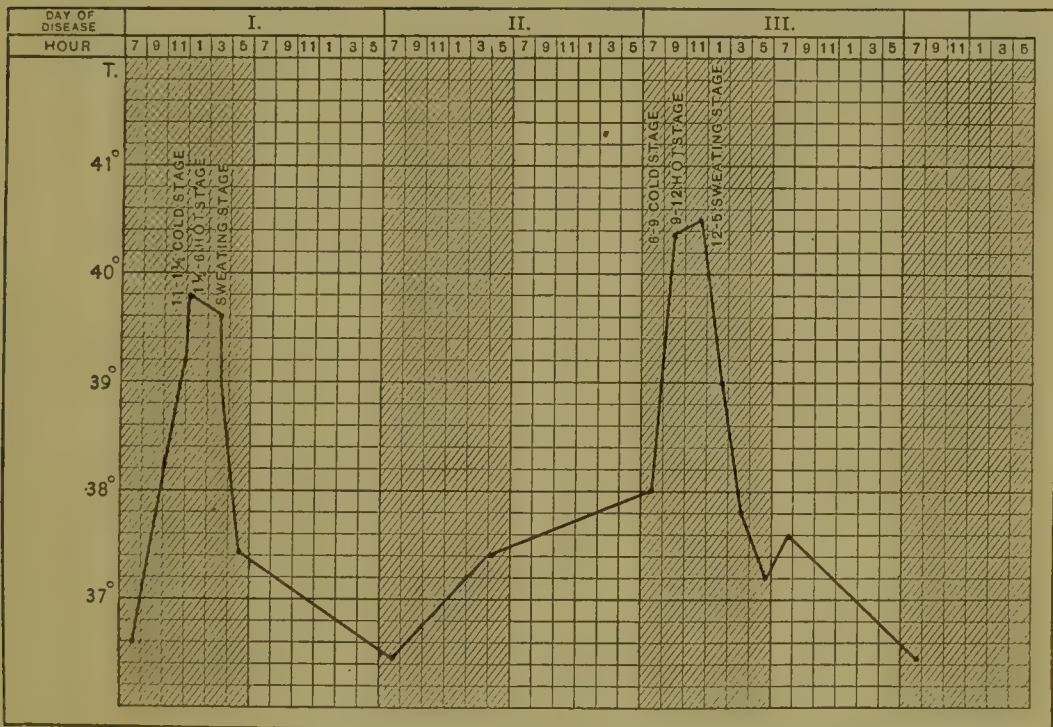


FIG. 12.—Temperature-curve in a case of tertian intermittent fever (Sahli).

and so on until the normal is reached. Usually, following the paroxysm, the temperature becomes subnormal (about  $97^{\circ}$  F.;  $36^{\circ}$  C.). The length of the typical malarial paroxysm ranges, in all save exceptional instances, from eight to twelve hours.

(b) *The Manner in which the Paroxysms Recur.*—The special characteristic of this form of intermittent is the regularity with which the paroxysms recur in cases that are not under treatment. The intermission, or time between two successive paroxysms, is most frequently twenty-four hours (quotidian intermittent fever); almost as often it is forty-eight hours (tertian intermittent); and less frequently it is seventy-two hours (quartan intermittent). If there be two paroxysms on one day—a rare occurrence—the term “double quotidian” is used to designate the case. Of the above types, as stated in the life-history of the parasite, two only—the *tertian* and the *quartan*—have been clearly



distinguished. The quotidian ague (the most frequent clinical variety) is generally due to double infection by the tertian parasite, and very rarely is it to be attributed to the presence in the blood of three groups of the quartan parasite, resulting in daily sporulation. It sometimes happens that the paroxysms recur a couple of hours later each successive day, when it is called a "*retarding*" intermittent fever, or they may recur a little earlier, when the term "*anticipating*" is employed.

*Other More or Less Characteristic Symptoms.*—Apart from the paroxysms and the regularity with which they recur, *splenic enlargement* is almost always present, and hence is of considerable clinical import. After the first paroxysm or two the swelling is usually marked and demonstrable, especially by palpation. The organ can be shown to increase in size with each succeeding paroxysm. Tenderness is elicited on pressure, and commonly outlasts the course of the affection for a considerable length of time. Moderate enlargement of the liver may be present, but this is neither so significant nor so constant as enlargement of the spleen.

Connected with the *skin* are two symptoms of considerable diagnostic value: (1) a *yellowish-brown discoloration*, the so-called "malarial complexion," due to the deposition of pigment; and (2) *herpes*. The latter occurs usually on the prolabia or on the nose, though rarely elsewhere. Other skin-eruptions, as urticaria, purpura, etc., have been described by authors, but they have no real clinical worth.

As stated under Pathology, *acute dilatation of the heart* may develop, attended with the usual physical signs of this condition, but it rarely lasts longer than the brief febrile paroxysm. Murmurs of functional origin may also be heard in the heart during the attack, and the *lungs* upon auscultation sometimes present the signs of a dry bronchitis.

The *urine* may contain a small amount of albumin during the pyrexial period, and rarely there is acute nephritis, but only in the severest types. There is a temporary increase in the amount of urea eliminated during the febrile movement, and this may be observed from two to six or eight hours before the chill, so that an approaching paroxysm can be foretold if a quantitative analysis of the urine be made at the proper time (Jaccoud). The increased proportion of urea in the urine is occasioned in the same manner as in fevers generally.

*Gastro-intestinal symptoms* may be present, but are not prominent, if we except a diarrhea which is sometimes considerable. *Catarrhal jaundice* may be observed, but this is limited to the graver forms of intermittent.

**Clinical Varieties.**—Besides the typical attacks, *mild* or *rudimentary* forms are met with, these either being due to slight infection or appearing as the remnant of cases of usual severity after active treatment. The separate stages of the febrile attacks are not well marked, and one or more may be missing; thus the chill may be absent (dumb ague), and less frequently the sweating stage may fail to appear.

In *children* there is no rigor noticeable. They grow pale, the visible mucous membranes often being slightly livid during the chill, and the paroxysms may be initiated by a convulsion or by other nervous phenomena.

(II.) **Pernicious Malarial Intermittent.**—This truly serious form

occurs chiefly in highly malarial districts, and rarely also in the widespread regions in which the simple variety prevails. Hence in the United States it is encountered most frequently in the Southern and Southwestern States. In this form of malaria the parasites of estivo-autumnal fever are constantly associated. The paroxysms do not recur with strict regularity, and the primary paroxysms are rarely pernicious in character; but second or subsequent attacks may, in addition to the usual symptoms, present the gravest phenomena.

**Pathology.**—This type of malaria may arise (1) as a fresh infection, and (2) as a reinfection.

(1) *Infection.*—The blood is more or less hydremic, and the blood-disks are in all stages of disintegration. The *spleen* is considerably swollen, soft, and its parenchyma is turbid and lake-colored, all its tissue elements being more than naturally pigmented, though this may not be macroscopically appreciable. Upon microscopic examination, however, pigment-granules and red corpuscles containing parasites and phagocytes are observed, particularly in the pulp adjacent to the arterioles. The *liver* is enlarged, soft, and turbid, and pigmentation occurs, but it is also microscopic. In the minute vessels phagocytes and parasites containing pigment are perceptible within the red corpuscles, and numerous small necrotic areas have been observed. The *kidneys* show microscopic pigmentation, most marked in the vicinity of its blood-supply. Minute areas of cell-death are sometimes seen. The *brain* may be abnormally colored, assuming in severe cases a chocolate tint, and in mild types a lighter hue. The brain-tissue is often anemic, and more rarely edematous. Occasionally there is congestion. The minute vessels and capillaries are literally blocked with phagocytes and blood-disks more or less disintegrated (containing parasites), and perivascular infiltration and minute hemorrhages may rarely occur, producing a focal lesion.

(2) *Reinfection.*—The blood is often extremely hydremic. The *spleen* may or may not be much enlarged, and is usually quite firm, with a well-marked pigmentation that is obvious to the naked eye. The *liver* is, as a rule, increased in size to a moderate extent only, and is somewhat indurated, while macroscopically it is seen to be deeply pigmented. The changes presented by the *kidneys* differ in no essential manner from those of the liver. The microscopic appearances of the liver, spleen, and kidneys, apart from the fact that the amount of pigment present is relatively greater, are entirely analogous to those met with when a fresh infection occurs.

**Clinical Varieties.**—Three varieties merit description:

(a) *Congestive Chills (Algid Form).*—These are accompanied by raging gastro-intestinal symptoms (vomiting, purging, etc.), inducing systemic collapse, which simulates to a nicety the algid stage of cholera. The temperature of the interior of the body is much elevated. True dysenteric symptoms may arise, and in a certain proportion of the cases jaundice, followed by grave nervous symptoms, may be a secondary development. This condition is to be discriminated from yellow fever, with which it has frequently been confounded. The parasites in this affection center in a special manner in the gastro-intestinal mucosa, in the vessels of which they may be seen in unusual numbers, sometimes



forming distinct thrombi. In the United States this is the most common among the pernicious forms.

(b) *Hematuric Pernicious Malaria*.—In this form the chill is severe and prolonged, and during the hot stage the urine is bloody and scanty, containing considerable albumin, with bloody epithelial and granular casts. Hemorrhages from other outlets of the body (mouth, rectum, vagina, nares, etc.) may also occur, together with larger and smaller cutaneous ecchymoses, and the yellowish-brown malarial complexion is intensified. The mind may remain clear throughout, although the patient is restless and anxious. Urinary suppression may ensue, and uremic toxemia be superadded; the greatest dangers being cardiac failure, uremia, and delirium (or coma independently of the latter). Death is rarely the direct consequence of excessive loss of blood.

(c) *Comatose Form*.—The chill may be absent. Grave cerebral symptoms, as acute delirium or sudden coma, seize the patient violently. The hot stage is attended with high fever, and if the patient survives the paroxysm, the violent nervous symptoms either disappear suddenly with the appearance of the sweating stage, or may outlast the latter by several hours. Primary paroxysms rarely prove fatal, but recurrences bring imminent danger. This dangerous variety is due to an inordinate localization of the malarial parasites in the brain, where they form complete thrombi, and induce, as a consequence, pathologic lesions in the adjacent structures.

(III.) **Remittent or Continued Malarial Fevers (Estivo-autumnal Fever)**.—On account of the intensity of the gastro-intestinal symptoms this variety is also termed *bilious remittent fever*. Its severity exceeds that of intermittent malarial fever. It prevails for the great part in warm and truly tropical climates, though it is also seen in its milder forms in temperate climates. The estivo-autumnal parasites previously described are the specific cause of the disease.

**Pathology**.—Melanosis of the spleen, liver, and brain is generally observed; on the other hand, in rare instances in which the specific parasite had even been demonstrated during life, the internal organs were found to be non-pigmented on autopsy. The degree of the pigmentation depends upon the length of time that the patient has been infected, as well as upon the frequency of reinfection. The *spleen*, if it be a fresh infection, becomes swollen, but is soft; in protracted cases it become permanently enlarged and firm. On microscopic examination the pigment is seen to be most abundant in the splenic pulp and within and around the splenic veins. The *liver* is enlarged in like manner. The pigment that is found in the form of granular masses in all the hepatic tissue elements (especially Kupffer's cells, vessels, vessel-walls, and perivascular tissue) gives to the organ a bronzed appearance ("bronze liver").

As in pernicious malaria, so in this affection, the *brain*, and particularly the gray matter, is in long-standing cases of a dark brown or almost black color. Here, again, most of the pigment is in and around the *arterioles*. The latter are often found stuffed with phagocytes and blood-disks which contain pigmented parasites. Punctate hemorrhages may occur in the brain. Other organs and tissues of the body, including the *lymphatic glands* and the *skin*, become more or less deeply pig-



mented. The *blood* shows marked hydremia, with partly or wholly degenerated red blood-disks in abundance.

**Symptoms.**—There may be prodromal symptoms, such as headache, anorexia, and epigastric oppression, lasting a day or two, but these signs are variable. There may be daily or bi-daily paroxysms of fever which resemble the ordinary quotidian and tertian intermittent forms, with this difference, however, that the febrile paroxysms are of longer duration (twenty hours or more). Both the rise at the onset and the decline at the end of the paroxysm are more gradual than in true intermittent malarial fever, and the initial chill may even be wholly absent. The febrile attacks are often “anticipating,” so that it may happen that the succeeding paroxysm will begin before the elevated temperature of the preceding touches the normal level, giving rise to a remittent type of fever which often exhibits considerable irregularity. The remissions may become shorter and shorter, producing finally a continued type of curve—continued malarial fever.

In *typical cases* of remittent fever a chill generally occurs at the onset, but is less severe than in malarial intermittents. Shortly after the chill the temperature rises rapidly, so that in ten or twelve hours it may reach 104° or 105° F. (40.5° C.). The *pulse* is full and accelerated to 100 or 120, and there is rending headache. Nausea and vomiting are common; oppression in the epigastrium is intense, and there is well-marked tenderness in the latter region. The *spleen* is found to be enlarged on palpation. *Nervous symptoms* (delirium, coma, etc.) may develop speedily, and rarely a mild bronchitis may also arise.

About midnight the remission in the temperature and sweating begin, in consequence of which the headache and gastric symptoms largely disappear. The temperature usually drops to 100° F. (37.7° C.) by the next morning, to be followed by a new exacerbation of fever, which commences about noon of the second day. The same symptoms now repeat themselves. The affection has usually, by this time, reached its acme, and the temperature may have risen to 106° F. (41.1° C.). Grave nervous symptoms may also have appeared. The urine is greatly diminished in amount or even suppressed; it is often slightly albuminous, sometimes even bloody, while either a slight hematogenous (?) or marked hepatogenous jaundice may appear. *Herpes labialis* is quite common. The nocturnal remission again ensues, and in the *mild types* or in those brought promptly under suitable treatment the febrile paroxysms grow briefer until the remittent is merged into an intermittent form of fever. The course of light cases is run, usually, within two weeks.

In *severe types* or in neglected cases the separate febrile paroxysms grow longer until the remissions become slight and simulate continued fevers. These are the cases that are distinguished by the same symptoms as those that mark typhoid fever, save the eruption which is peculiar to the latter. The discovery of Laveran is of the highest practical value in this category of cases. The course of the attack, if not properly treated, generally prolongs itself to three, four, or more weeks, and under these circumstances the salient features of pernicious intermittent may suddenly appear and the disease may terminate life. On the other hand, *mild forms*, in which the fever is of the continued type, also occur, and these yield promptly to the specific—quinin.

(IV.) **Malarial Cachexia.**—This is an exceedingly chronic condition, and is usually a remnant of one of the acute forms, particularly of the ordinary intermittents. When the latter are not properly treated, they are apt to drag on, and finally assume the characteristic features of chronic malarial cachexia. The condition may, however, develop in truly malarial localities without the intervention of primary acute malaria. It originates, however, only in truly malarial districts.

The **symptoms** are varied both in character and in intensity. There is fever at intervals, but chills do not occur, and the temperature-curve is typical neither of remittent nor intermittent fever, but may approximate either the one or the other. Again, the fever is sometimes wholly irregular, though its range is not high, and it seldom excels  $103^{\circ}$  F. ( $39.4^{\circ}$  C.). The *skin* often presents the dirty yellowish-brown complexion to a marked degree. The spleen is enormously enlarged and indurated, and hypertrophy with hardening of the liver may also be pronounced. The *blood* is profoundly anemic, the count in one of my own cases showing but 1,300,000 red corpuscles per cubic millimeter.

Many of the local and general symptoms that remain to be given (including, in part, the fever) are chiefly dependent upon the well-marked anemia. Among general features may be mentioned debility, frequent sweatings, and dropsy. *Nervous symptoms* may also be noticeable, and chief among these are tremors, neuralgia, palsies, vertigo, wakefulness, and nervous palpitation of the heart. Among the rarest concomitants of this condition is *paraplegia*. Slight cough and dyspnea evidence the presence of mild bronchitis; and anorexia, nausea, diarrhea, and other symptoms mark the presence of chronic gastrointestinal catarrh. The joints and voluntary muscles may be painful. Hemorrhages from the various mucous surfaces and into the retina are common; and I have seen one case in which spongy, easily-bleeding gums, with cutaneous ecchymoses and numerous petechiæ, pointed to the existence of associated scorbutus. Tuberculosis finally developed and carried off the patient. Not only the latter affection, but also chronic dysentery, chronic Bright's disease, and amyloid disease, may develop and prove serious complications. These cases do well, generally, if the patient can be removed permanently from the malarial district and if proper treatment be persistently pursued. In long-standing cases the spleen does not return to its natural dimensions. In all other instances, however, complete recovery may be expected, though it may require months or even years to bring it about.

(V.) **Masked Intermittent.**—This presents itself in much the same forms as chronic malarial cachexia, but with the important difference that there is no fever. This type comprises a long list of conditions, at the head of which stands *neuralgia*, most frequently involving the supra-orbital branch of the trigeminus. Often a striking periodicity is observed, the painful paroxysms usually beginning in the morning and terminating in the late afternoon hours, the patient's sufferings increasing steadily in intensity until just before the close of the attack, when they suddenly abate. Among other nerves implicated with relative frequency are the occipital, the intercostals, and the sciatic. Except the blood-appearances be characteristic or unless the attacks yield promptly to quinin, a certain diagnosis of malarial neuralgia should not be ventured.



Masked intermittents may assume the forms of paresthesia, anesthesia, convulsions, or paralysis; non-febrile intermittent malaria may also appear under the guise of edema, hemorrhages from the various mucous outlets of the body or into the skin, intestinal flux (diarrhea, dysentery), dyspepsia, etc. But, since these affections may all obey the law of periodicity, caution should be used in pronouncing in favor of malarial infection. Indeed, unless they yield readily to the therapeutic specific, a positive statement had better be withheld.

(VI.) **Malarial Hematuria and Hemoglobinuria.**—I have previously described a hemorrhagic form of pernicious intermittent in many cases of which hematuria is a prominent symptom. Among other general features are jaundice, prostration, nervous symptoms, and nephritis (Plehn). The blood shows non-pigmented parasites (forming rosettes) and sometimes crescents and pigmented leukocytes.

Boisson<sup>1</sup> in 3 cases of hemoglobinuric fever, occurring in soldiers attacked with malaria in Madagascar, found an enormous reduction in the red corpuscles, reaching 670,000 in 1 case, while 7 out of 10 red cells contained parasites. I have observed several instances of malarial hematuria in the Kensington district of Philadelphia, and find that they are met with wherever the moderate forms of malaria prevail.

The **symptoms** consist of a mild cold stage, a subfebrile temperature to which is added hematuria, or more often hemoglobinuria. The paroxysms may recur daily, bi-daily, or at longer intervals, and in severe forms the hemoglobinuria may be continuous, with aggravations at definite intervals. The *diagnosis* demands the demonstration of the malarial parasites in the blood, and of the hemoglobin in the urine. Tyson recommends Teichmann's (hemin crystals) test to show the presence of hemoglobin. The earthy phosphates are precipitated, filtered out, and a small portion placed on a glass slide and carefully warmed until completely dry. A minute granule of common salt is carried on the point of a knife to the dried mass and thoroughly mixed with it. Any excess of salt is then removed, the mixture is covered with a thin glass cover, a hair interposed, and a drop or two of glacial acetic acid allowed to pass under. The slide is then carefully warmed until bubbles begin to make their appearance. After cooling, hemin crystals can be seen by the aid of the microscope, and, though often very small and incompletely crystallized, are easily recognizable by an amplification of 300 diameters. Chemically they are hydrochlorate of hematin.

**Complications.**—The author's analysis of 1780 cases of malaria showed complications in about 10 per cent. The more common among these were not particularly grave in nature, as may be seen by a glance at the subjoined list, in which they are placed in the order of frequency of occurrence: Enteritis (16), nephritis (14), rheumatism (10), typhoid fever (8), lobar pneumonia (5), jaundice (5), and dysentery (4). The opinion of the profession is, and long has been, divided upon the question, "Has pneumonia any special connection with malaria?" but, according to the results of my own collective investigations, *lobar pneumonia* cannot be regarded as being frequently in association with the latter disease. That it is so rarely, however, cannot be denied, since the diagnosis in two of the cases was confirmed by autopsy.

<sup>1</sup> *Rev. de Méd.*, May 10, 1896.



*Typhoid fever* is a complication of malaria, according to these researches, but the relationship between these two leading affections cannot be close.

**Diagnosis.**—(1) **Of Intermittents.**—This is quite difficult, unless the brief febrile paroxysms, with their characteristic stages and other more or less diagnostic features (enlarged spleen, malarial complexion, and herpes), together with the rigid periodicity of the paroxysms, be present. The diagnosis is assisted by a knowledge of the fact that the patient resides in a malarial district. In cases in which a microscopic examination of the blood cannot be made early a positive diagnosis is rarely possible until after the patient has been observed long enough to ascertain the manner in which the paroxysms recur, in addition to noting the symptoms presented. The only unquestionable method of diagnosis is by means of a microscopic examination of the blood, which will show the tertian or quartan parasite (*vide infra*).

**Differential Diagnosis.**—Non-malarial affections, exhibiting an intermittent form of fever, are often mistaken for malarial intermittents. Of these (a) *pyemia* is very apt to be thus confounded, owing to the fact that it may present a similar temperature-range. It will be observed, however, that the chills recur at more irregular intervals, and in this disease the more profound prostration and other general features during the intervals between the febrile exacerbations serve to distinguish it from intermittent malarial fevers. The etiologic factors and place of residence are also to be considered. In doubtful instances every effort should be made to examine the blood microscopically, and, if this be impossible, the therapeutic test will, as a rule, remove the doubt. *Leukocytosis* is common in pyemia and absent in malaria.

(b) *Acute tuberculosis* and, more rarely, *incipient chronic tuberculosis* may present a febrile movement in no way differing from quotidian intermittent, except that in the former the pyrexia develops in the afternoon, instead of the forenoon, as in the latter. A clear history, the associated local and general symptoms, along with the results of a careful physical examination, usually render tuberculosis probable and distinguish it from malarial intermittents. Leukocytosis is common in tuberculosis and is absent in malaria, while in tuberculosis the chills recur despite the use of quinin, and this is not the case in malaria.

(c) *Ulcerative endocarditis* may exhibit an intermittent pyrexia, but in this affection the history is different, and the associated clinical features are more numerous and, as a rule, decidedly more grave. In endeavoring to eliminate a disease of so serious a character as ulcerative endocarditis, when the symptoms are strongly suspicious of the latter a blood-examination should be made without delay. The irregular forms of intermittents are difficult in the extreme to diagnosticate. If, in suspected cases of "erratic" malaria, quinin is resisted, we cannot feel certain of our diagnosis unless we obtain the microscopic evidence of the presence of the malarial parasite in the blood.

(2) The diagnosis of **remittent fever** would be easily made if it did not sometimes bear a strong resemblance to typhoid fever. On account of this fact its certain diagnosis demands the detection in the blood of the estivo-autumnal parasite. This, at first, is a small hyaline, motile body with little pigment, but in cases lasting a week or more assumes the oval

or crescentic shape with much pigment. In *typhoid fever* the history points to a more gradual onset, the remissions are less marked, and there is not the epigastric oppression witnessed in remittent fever. Again, typhoid has its characteristic eruption.

Remittent fever must not be confounded with typho-malaria, nor with continued thermic fever (Guitéras). The following will be found a ready and efficient method of examining the blood: The finger or lobe of the ear should be carefully cleansed, and then slightly cut with a sharp lancet. The first drop of blood should be wiped away and the second collected on the center of a clean cover-glass, which is immediately placed upon a clean slide and the blood allowed to spread in a thin film. The examination should be made with an oil-immersion objective. If desirable to preserve the specimen or if impossible to make the microscopic examination at once, smears should be prepared by laying another cover upon the first, allowing the blood to spread in a thin layer, and then sliding them apart quickly and drying in the air. The specimen may then be fixed in a mixture of equal parts of alcohol and ether by heat or one of the other usual methods. The most satisfactory stain is methylene blue. A few drops of a watery solution should be placed upon the cover-glass, allowed to remain about a minute, and washed off with clean water. The specimen can be examined in water or dried and mounted in Canada balsam. The organisms appear as small blue bodies, often containing pigment. Eosin may be used as a counter-stain. For the crescent and oval forms, which are sometimes difficult to find, it may be advantageous to allow a drop of blood to dry upon the cover-glass without spreading, fix as before, and then wash with dilute acetic acid; wash thoroughly with water and stain as before. The red cells are dissolved, and only the white cells and the parasites remain upon the slide.

**Prognosis.**—All cases of uncomplicated intermittent fever under prompt and proper treatment will probably recover, though fatalities sometimes occur. It is to be borne distinctly in mind that in certain malarious regions and in certain seasons pernicious types are prevalent, but, since these arise only after one or more preceding mild attacks, they are preventable. *Primary pernicious attacks* are moderately dangerous, while recurrences are highly so. The mortality-rate in this variety of malaria is between 20 and 25 per cent., and simple intermittent fever may, if not checked, suddenly develop into the most malignant type and result fatally.

In *remittent fever* a fatal issue may be due to asthenia, particularly when the type is severe and when, following the typhoid state, wrong notions as to treatment prevail. The severity of the infection may be estimated by appreciating the degree of fever and the severity of the nervous symptoms. Suppression of urine, followed by uremic symptoms, hemorrhages, and intense jaundice, are the chief untoward complications.

**Treatment.**—1. For **intermittent malarial fever** there is an almost infallible remedy in quinin. "When shall its use be commenced?" is a pertinent question. It would certainly seem highly desirable to check the course of the disease as soon as possible, and especially since transmission of the simple intermittents into the pernicious forms may occur if the disease be not arrested. At the present day specific treatment is



often delayed in order to give full opportunity for making a blood-examination with a view to completing the diagnosis. There is no decided advantage in commencing the use of quinin during the first paroxysm, when the blood may be examined; but on finding the case to be one of malaria, quinin should be administered after the paroxysm, so as to prevent a recurrence. For like reason, if the history at the physician's first visit, combined with the symptoms presented, make the diagnosis of intermittent malaria reasonably certain, and there is no opportunity to examine the blood microscopically, the principal antiperiodic remedy should be commenced at the close of the paroxysm, especially if the patient be living in an infected district.

The quinin cures malaria by acting directly upon the intracorpuseular hematozoa.

During the *paroxysm* we should aim to make the patient comfortable. He is to remain in bed, is to be well covered, and external heat applied during the cold stage; and he is to be lightly covered, given cooling drinks and cold spongings during the hot stage.

During the *apyrexial intervals* the patient may leave his bed, provided that he feel strong enough, and, as before intimated, the specific remedy is given during the afebrile period. Certain authors recommend that the entire daily quantity be given at one dose from four to six hours before the succeeding paroxysm is expected, the object being to surcharge the blood at the time when the hematozoa sporulate. Others give the remedy in divided portions, administering the last dose from four to six hours before the next paroxysm is due. It may matter little which of these two methods is pursued, yet my own experience leads me to favor the divided doses rather than the single large ones. The total amount per day required to cut short the intermittents is from 16 to 20 grains (1.036 to 1.296) in most temperate climates. When this fails more may be given—24 to 30 grains (1.555–1.944). My own practice has been to administer immediately after the close of the sweating stage gr. iv or v (0.259 or 0.324), repeating the same dose a few hours later, and the remaining 8 or 10 grains (0.518 or 0.648) (or one-half the daily dose) six hours before the time for the next paroxysm. I have thus escaped the slight toxic symptoms (tinnitus, deafness, nausea, etc.) which are apt to follow single large doses. The remedy is best given in capsules, followed by a few drops of dilute hydrochloric acid, with a view to dissolving the quinin in the stomach. After the attacks cease to recur quinin should be continued in amounts of 6 to 8 grains (0.388 to 0.518) daily for several days. If quinin cannot be taken *per oram*, it may be tried by enema or by suppositories in appropriately large doses. Rectal irritability may thus be produced, yet in very young subjects, who cannot be induced to swallow capsules, I have for a long time administered quinin by suppository.

The physiologic effects of the drug can be quickly obtained by administering it hypodermically. Hence, if there be no time for absorption from the stomach (four hours being the shortest period it is safe to allow), the drug should be thus employed. For this purpose the more soluble salts (hydrobromate, etc.) of quinin are to be preferred to the ordinary and more insoluble sulphate, which requires the addition of a mineral acid.



Many preparations of cinchona other than the salts of quinin may be tried, and among these cinchonin administered in the same manner as the latter is the best substitute. Some contend that the sulphate of quinidin has antiperiodic power, almost equal to quinin. In prolonged cases the salts of quinin and other preparations of cinchona sometimes lose their specific influence, and arsenic is then to be employed, either alone or in combination with the former agents. The dose of the arsenic, beginning with  $\mathfrak{M}\text{iv}$  (0.266) t. i. d. of Fowler's solution, must be slowly increased until its full physiologic effects are produced. Arsenious acid often does better service than Fowler's solution, although it has to be administered in augmenting doses to the amount finally of gr.  $\frac{1}{4}$  (0.0162) daily. So soon as the disease is controlled the dose of arsenic is to be greatly reduced, but the drug is not to be altogether withdrawn for several days. Administered as above indicated, this remedy is most efficacious in malarial cachexia and masked forms of intermittents, and should in the latter conditions be combined with iron and quinin. While in charge of the out-patient service of the Episcopal Hospital, Philadelphia, I employed in chronic malarial cachexia, with very satisfactory results, the sulphate of cinchonidin in daily doses of gr. xxx-xl (1.944-2.592). In this class of cases Warburg's tincture ( $\mathfrak{z}\text{ss}$  (16.0) three times a day) has been warmly recommended.

**2. The Treatment of Pernicious Intermittents.**—(a) *Prophylaxis.*—By treating all ordinary intermittents actively after the first paroxysms the occurrence of pernicious forms can be obviated. Not to pursue this course in seasons and in localities in which these serious types are known to prevail, but to delay for second and third paroxysms in order to be able to study the blood, is criminal.

(b) *The first pernicious attack* must be treated immediately, and there is not a moment to be lost. Hence in all varieties of pernicious intermittents quinin should be administered hypodermically until the patient is fully cinchonized—a condition that must then be maintained for several days. In all varieties stimulants are to be used freely if the heart's action becomes feeble, and the patient is to be well nourished throughout. There are other details, though of relatively minor importance, and they vary with the individual forms. Thus in "congestive chills" external warmth is useful, and morphin combined with atropin should be given hypodermically, this combination tending to allay gastro-intestinal symptoms as well as to warm the extremities, and meeting really important indications. Rectal feeding must be resorted to should the stomach refuse to retain nourishment. In the *comatose form* the nervous symptoms are most successfully combated by prompt and energetic antiperiodic treatment, together with vigorous stimulation and feeding, since they are not due to cerebral congestion, but to the intensity of the infectious process.

(c) During the apyrexial period every effort must be made to prevent a recurrence of the paroxysm, and to this end the patient must be kept fully cinchonized until the time for the next paroxysm is over, and then be removed from the malarial to a non-malarial district.

**3. Treatment of Remittent Fever.**—The mode of treatment in this form differs somewhat from that appropriate for intermittents. At the onset a mild mercurial is advantageous (calomel gr.  $\frac{1}{4}$  (0.0162) every hour for

three doses), followed by a saline laxative (Rochelle salts,  $\text{ʒij}$ ; 8.0). During the febrile exacerbations cool spongings of the body, together with the use of the ice-cap, are serviceable. The gastric symptoms demand chipped ice by the mouth or small doses of cocain, and a mustard plaster externally. Immediately after the first remission sets in quinin must be exhibited, and large doses are now indicated (gr. xv (0.972), to be repeated at 8 or 9 A. M.). A third and even a fourth dose of the same size may be required. The exacerbations of fever generally yield to this remedy, but if, as rarely happens, they do not, then small doses of pilocarpin (gr.  $\frac{1}{8}$  to  $\frac{1}{6}$ ; 0.008 to 0.010) may be administered hypodermically during the height of the fever. This causes free sweating in many instances, and in consequence renders the remission more marked and more prolonged; thus, in short, rendering the course of the affection speedily favorable. The heart, however, must be carefully guarded when this depressing agent is prescribed.

A case that has been allowed to run on for one, two, or more weeks is often greatly benefited by the use of Warburg's tincture, as before recommended, for several days, when quinin may be re-employed. The patient, especially if the case be protracted, must be vigorously fed, and *per rectum* if it cannot be accomplished by the mouth. In typical cases, which are promptly controlled by quinin, stimulants are rarely needed, or at least not until the convalescent stage is arrived at. In severe and neglected cases the indications for their employment may be presented early, and they should then be given, the physician conforming to the same rules as in typhoid and other acute infectious diseases. The renal congestion and anuria are to be met by internal diaphoretics (pilocarpin, etc.) and by saline laxatives. Most efficacious, perhaps, is a combined hot-water and steam bath. The patient is placed in hot water, and then a blanket is put around the neck, its free ends being allowed to extend over the edges of the tub. This may be repeated, if necessary.

**4. Treatment of Malarial Hematuria.**—The treatment of hematuria as a symptom of grave types has been embraced in the treatment of hemorrhagic pernicious malaria. The use of quinin in moderate doses (gr. xvj—1.036—daily) successfully relieves the hemoglobinuria occurring in connection with mild forms of malaria, and its subsequent use in smaller doses (gr. iij (0.194) to gr. vj (0.388) daily) will prevent a recurrence. It is claimed by some writers that quinin may produce hematuria (Plehn, Richardson, and others), and also that this remedy is of no value in combating this symptom. The specific remedy should not be abandoned, however, and large quantities, such as might act as an irritant to the renal tissues, are not necessary to effect a cure, except in pernicious forms.

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## DYSENTERY.

**Definition.**—An infectious inflammatory disease of the large intestine, characterized anatomically by ulceration of the intestinal mucosa, and clinically by frequent mucous and bloody discharges, tenesmus,



fever and prostration becoming profound, a tendency to abscess-formation in the portal system, to paralysis, and, finally, to pronounced anemia. It is a truly epidemic disease, yet it also occurs constantly in endemic form, and particularly does this occur in temperate climates.

**Historic Note.**—Few diseases have been longer known than dysentery, of which we have a description by Hippocrates. Galen localized the chief seat of the affection in the colon, and in 1626, Sennertus defined its sporadic and epidemic character and some of its leading clinical features. To Morgagni belongs the credit of having made the first postmortem anatomic study of the disease. Further and more accurate pathologic contributions were made in the earlier part of the present century by Cruveilhier and Rokitsansky, and, more recently still, the whole subject of the morbid anatomy of this disease has been carefully investigated by Virchow, whose results have settled most of the questions connected with the subject. In the United States dysentery has prevailed epidemically upward of a century, the time of greatest prevalence in different districts having been about the middle part of the present century (1847–55). Woodward has given us the only complete record of the various outbreaks in this country, and an account of the ravages of dysentery in both armies during the War of the Rebellion is given in his *Report*, which records 259,071 cases of acute and 28,451 of chronic dysentery. The disease is far less frequent than formerly, owing to the advance made in recent times in sanitary science, in consequence of which some of the predisposing conditions have been overcome.

**Etiology.**—As stated below, there are three distinct clinical types of the affection, each of which has special etiologic factors. A few general etiologic considerations, having reference to the different forms in common, may be adduced here, and, as each variety presents different anatomic lesions in leading particulars, their pathology will be considered separately.

Among disposing factors, *season* heads the list, dysentery being most common in the summer and autumn; great and sudden changes of temperature are more potent than equal changes in humidity. *Climate* has a marked effect, and high temperature must be regarded as a powerful agency, since the disease is much more prevalent in warm than in cold climates, though it is met with in epidemic form as far north as Norway. *Malarial districts* suffer more than non-malarial. This may be due to the fact that an attack of malaria may leave the body of the sufferer more receptive to the specific poison of dysentery, or the external conditions which favor the development of the plasmodia may also favor the growth of the dysenteric poison. The latter view gains some support from the well-known fact that water taken from stagnant pools in marshy localities (in which malaria is apt to prevail) may give rise to the symptoms of dysentery. *Unhygienic conditions*, as shown by the local epidemic outbreaks in jails, barracks, institutions, etc., predispose to the affection.

Among factors connected with the individual are (*a*) *catarrhal* conditions of the intestinal tract, particularly if the latter be caused by unripe fruit or other unwholesome forms of food; (*b*) *Age*. Although no age enjoys immunity against dysentery, most cases are met with in



adults under thirty-five years. *Sex* and *race* are probably without appreciable influence.

### CATARRHAL DYSENTERY.

**Pathology.**—There are two forms: (a) In this the solitary follicles are affected chiefly, and are the seat of hyperplasia, followed by necrosis, with the formation of small ulcers. This is common in children. (b) Here a purulent inflammation of the entire mucosa, with more or less erosion of the surface and superficial ulceration, exists. In both forms the lesions are mainly confined to the large intestine, though the ileum is sometimes implicated to a lesser extent.

**Special Etiology.**—The specific bacillus of catarrhal dysentery is not known to a certainty. It is probable, however, that it will be shown to be the bacillus coli communis, which may become pathogenic when the state of the mucous membrane of the bowel is altered by sudden changes of temperature, etc. (Arnaud, Maurel).

**Clinical History.**—There may be prodromes, lasting one or two days, which take the form of a mild gastro-intestinal disorder (anorexia, slight pains in the abdomen, followed by diarrhea).

The characteristic symptoms are mild colicky pains in the abdomen, followed by discharges from the bowel, which at first number from three to six daily. Soon they become frequent and are accompanied by straining and tenesmus, and now their number ranges from ten to no less than one hundred or more per day. Indeed, the desire to go to stool may be almost constant, and the rectum is the seat of intense burning sensations during and after each evacuation of the bowel. The character of the discharges varies with the different periods of the affection. During the first thirty-six or forty-eight hours they are feculent (sometimes scybalous masses), rather copious, and intermingled with some mucus and blood. For the next four or five days the stools are scanty, measuring from 2 drams (8.0) to  $\frac{1}{2}$  ounce (16.0), and are made up of a sero-mucous fluid or of a muco-purulent material with blood. The chief constituents of the stools are mucus, blood, and pus, any one of which may preponderate, thus giving rise to mucous (most frequently), purulent, or bloody stools.

Microscopic examination of the usually glairy stools shows red blood-corpuscles, numerous leukocytes, generally large, oval or round epithelioid cells containing fat-globules, vacuoles, and bacteria (especially those of putrefaction). Occasionally the *Cercomonas intestinalis* is seen (Osler).

A few shreds (portions of necrosed mucous membrane) may appear from time to time in the discharges, and particularly in severe forms of the affection. These usually increase in number at the close of the first week, and a little later the discharges become less frequent and the amount of mucus and blood diminishes. The stools are now of a greasy brown or dark-green appearance, fecal matter reappearing in them, and soon they are again fully formed.

**Other Symptoms Referable to the Alimentary Tract.**—The tongue has a greasy coating—moist at first, dry later—and at last may become red and glazed. Anorexia is present, with excessive thirst, and vomit-

ing may rarely occur. A distressing though uncommon symptom is hiccough. There will usually be tenderness over the line of the colon, but there is an absence of tympanites, and the abdomen is apt to be flat and somewhat tense.

The general symptoms are well marked in the severer types. The patient is much debilitated, sometimes even collapsed, as shown by the small, frequent pulse, cool skin-surface, the rapid wasting, and weak, hoarse voice. The temperature is not much elevated, though it may touch 103° or 104° F. (40° C.) at the outset, and the curve is an irregularly remittent one.

**Diagnosis.**—This can easily be made upon the intestinal features and from the character of the stools—frequent, small, slimy (or bloody) discharges, accompanied by distressing tenesmus.

**Differential Diagnosis.**—Symptoms simulating dysentery may appear in the course of certain rectal affections, such as strangulated hemorrhoids, syphilis, and epithelioma. In these conditions, however, there is a different history and the symptoms of proctitis are usually less acute, while a physical examination of the rectum will settle the diagnosis in doubtful cases.

**Prognosis.**—The *duration* of mild cases is from eight to ten days, and in severe types from three to four weeks. The prognosis varies in different epidemics or according to the type of the affection; but commonly this is not aggravated and recovery is generally to be expected. Occasionally, however, a dangerous condition arises. Besides the profound prostration and circulatory collapse above referred to, serious nervous symptoms (great restlessness, delirium followed by coma) may develop and cause a fatal termination. When death occurs it is usually due to exhaustion, and is seen particularly in persons previously enfeebled or in the aged. Complications influencing the prognosis are exceptional, though peritonitis and liver-abscess may rarely appear.

#### AMEBIC DYSENTERY (TROPICAL DYSENTERY).

**Bacteriology.**—This form of dysentery is caused by the *amæba coli* or the *amæba dysenteriae* (Councilman and Lafleur). The *amæba dysenteriae* is a unicellular, motile organism, in size 3 to 7 times the diameter of a red blood-corpuscle (15 to 30 micromillimeters). Its protoplasm consists of two zones—an outer colorless (ectosarc) and an inner granular zone (endosarc), with a visible nucleus and one or more vacuoles. This micro-organism was first described by Lambl (1859), but it remained for Lösch, and especially Kartulis, to show its close association with dysentery. It is now generally held to be the specific cause of tropical dysentery. The ameba (*amæba coli mitis*) is occasionally found in healthy individuals, and also in other bowel-affections than dysentery (mucous enteritis, simple diarrhea, proctitis due to engorgement), and two species are recognized—a virulent and a benign form (Quincke and Roos). The ameba is found not only in the discharges, but also in the pus from the secondary liver-abscesses.

Hehir has found the *bacillus dysentericus* associated with the *amæba coli*, and considers it to be pathogenic. He describes it as a short, straight bacillus, usually of a length about equal to one-third the diameter of a

red blood-corpuscle, with rounded ends, sometimes jointed, rarely curved. Later investigations, however, have not entirely confirmed the claims of this observer.

The mode of transference of the ameba is not definitely known, though the chief source of the dysenteric germs is most probably the drinking-water. The poison is feebly communicable by contact.

Amebic dysentery is not confined to the tropics, but is met with also, though less frequently, throughout Europe and North America.

**Pathology.**—The lesions are almost always situated in the large intestine, though rarely the ileum may be invaded. The first visible change is a hyperemia of the mucosa, most marked in the descending colon and rectum; but the changes which produce the characteristic dysenteric ulcer begin with infiltration and swelling of the submucosa, followed by necrosis, which involves the overlying mucosa with its epithelium (Kruse and Pasquale). How the amebæ reach the submucosa has not yet been observed. The visible infiltration occurs usually in circumscribed areas which are oval or hemispheric in shape, and project above the level of the surrounding mucosa. The submucosa presents a grayish-yellow appearance, and is soon thrown off in the form of a slough.

The ulcers take various shapes—chiefly irregular, and less frequently round or oval. Their edges are ragged and undermined, and the floor, which is more or less covered with pultaceous material, is rough or crater-like, and formed by the muscularis or the outer serous coat of the intestine. From the manner in which the ulcers are formed it is obvious that cellular infiltration (followed by necrosis) may occupy the submucosa for a greater or less distance beyond the borders of the ulcers. In this way fistulous channels may be produced beneath the mucosa and connect two or more ulcers. Usually this ulcerative process affects only certain portions of the large gut, especially the flexures—hepatic and sigmoid—and the rectum; but it may be general, and I have seen one instance of this kind. Similar cases are not uncommon in which the ulcers are so numerous as to include almost the entire mucosa of the large intestine.

*Healing* is attended with the development of fibrous tissue along the edges and in the base of the ulcer, and secondary contraction of this new connective tissue is often productive of colonic stricture, which is usually either partial or irregular. The cases that come to autopsy often show diphtheritic inflammation as a secondary or terminal condition.

The *microscope* reveals proliferation of the fixed connective-tissue cells, and the presence of amebæ in the walls and the base of the ulcers, in the lymph-spaces, and rarely in the blood-vessels. Pus can only occasionally be detected.

The *liver* may be the seat of prominent lesions. These are (*a*) *abscesses*, which may be single or multiple, the latter being small; and the former often large. The single or solitary abscess is usually situated near either the upper convex or the lower concave surface, while the abscess-cavity is formed in a manner similar to the intestinal ulcers. The area affected is at first infiltrated; it then becomes necrotic, and finally more or less liquefied. Upon the full development of the first stage the part invaded is a grayish-yellow pultaceous mass, but in the second or



necrotic stage the abscess contains a yellowish or greenish-yellow, spongy material with beginning liquefaction. The contents of the mature abscess consist of a greenish- or reddish-yellow purulent material and of remnants of liver-tissue. The walls of the recent abscess are irregular and ragged, those of an old abscess being dense and fibrous, and a section of the abscess-wall shows an inner necrotic zone, a middle zone (in which there is great proliferation of the connective-tissue cells, compression and atrophy of the liver-cells), and an outer zone of intense hyperemia (Osler). The contents of the abscess show either few or many amebæ, and only rarely pus. When pus is present it is due to a secondary infection by the pyogenic germs. In what way the amebæ gain access to the liver is not definitely known, but it is probable that in multiple abscesses they are propagated along the blood-current, either from the ulcers or from a single primary focus. Cultures are generally sterile.

(b) The parenchyma of the liver may be the seat of numerous circumscribed necrotic spots, which are supposed to be due to the action of the chemical secretions of the amebæ.

The **lungs** sometimes show changes similar to those in the liver, which are the result of direct extension of the hepatic abscess through the diaphragm into the lower lobe of the right lung.

**Clinical History.**—The mode of onset is variable except in a small proportion of the cases, in which it is sudden with well-marked symptoms. When, as generally happens, it is insidious, the initial symptom is often a trivial diarrhea. The affection is then characterized chiefly by intermissions and more or less marked exacerbations of diarrhea, the liquid stools containing necrotic tissue of a grayish-brown and sometimes yellowish-gray color. The latter are often bloody and mucoid, particularly at the outset, and in fully developed cases are fluid. The number of discharges per day is exceedingly variable in different epidemics, and even in individual cases, though in most instances they range from six to eight or ten daily.

*Microscopic examination* of the feces during the exacerbations discloses amebæ that are almost invariably endowed with motion, though usually not when the stools have become formed. Tenesmus is not a prominent feature in most cases, and may be entirely absent. Colicky abdominal pains are rare, and nausea and vomiting are equally so.

**General Symptoms.**—The febrile movement is usually present, but it is slight and exhibits marked variations. In certain instances, however, the temperature is below the normal curve throughout the entire or greater part of the course. From the time of onset there is gradual though progressive loss of flesh and strength, and anemia usually becomes well marked.

**Complications.**—The most common is *hepatic abscess*, and secondary to the latter may arise abscess of the right lung. Authors are not agreed as to the frequency of occurrence of liver-abscess<sup>1</sup> in amebic dysentery, but it is certainly comparatively rare in this country, not exceeding, perhaps, 3 per cent. of the cases. *Peritonitis* may result from perforation of a dysenteric ulcer, causing death. The point of per-

<sup>1</sup> For the diagnosis of this condition the reader is referred to the section on *Hepatic Abscess* in the article on *Diseases of the Liver*.

foration may, however, be in the rectum, when *periproctitis* is the result; or it may be in the cecum, when *perityphlitis* is the sequel. In tropical or subtropical countries the disease is often complicated with malarial affections, and in malarial regions intermittent and remittent fevers are among the commoner complications. The presence of an intermittent fever is not, however, sufficient to warrant the assumption that malaria complicates dysentery; and in order to show the latter combination we must be able to demonstrate the presence of the *plasmodium malarie*. In pyemia and in suppurative processes generally—conditions sometimes met with in dysentery—the temperature-curve is often distinctly intermittent. *Typhoid fever* is a rare complication. The latter disease cannot be said to coexist with dysentery unless all of the characteristic symptoms are present. Certain cases of dysentery are characterized by the development of the typhoid state, and pyemia and septico-pyemia may appear late. Among special manifestations of the latter are pylephlebitis, pericarditis, endocarditis, pleuritis, and rheumatoid pains in the joints.

**Diagnosis.**—The slow course, marked by intermissions and exacerbations of bloody fluid stools, the mild general symptoms, apart from emaciation and debility, are characteristic features, but a positive recognition of the affection demands a microscopic examination of the stools. Cases have been recorded by Councilman and Lafleur in which the diagnosis rested upon amebæ being found in the sputa, the latter being complicated with pulmonary and hepatic abscesses which discharged through a bronchus, while the intestinal symptoms were negative.

**Prognosis.**—The prognosis is graver than in the catarrhal variety, and the mortality-rate in certain epidemics has been frightful, particularly among soldiers in the field (amounting to 70 or even 80 per cent.). In sporadic cases the danger to life is less, the mortality-rate in temperate climates being not over 5 or 6 per cent. The complications which render the prognosis unfavorable are various (peritonitis, hepatic and pulmonary abscess, pyemia secondary to the latter, broncho-pneumonia, malaria, etc.); death may be due to hemorrhage or peritonitis, but in a preponderating proportion of the cases to asthenia. A dangerous degree of debility is indicated by great nervous depression; a cool, clammy skin; a sunken, pinched facies; a dry tongue; a feeble, rapid pulse; and by restlessness, alternating with marked apathy or low muttering delirium.

**Course and Duration.**—The average duration ranges from eight to ten weeks in uncomplicated cases; the disease can, however, be cut short by appropriate treatment. It manifests an innate tendency to pursue a chronic course, interrupted by frequent exacerbations or true relapses, and convalescence occupies a long period of time in consequence of the marked anemia and debility which ensue.

#### DIPHTHERITIC DYSENTERY.

This is an intestinal inflammation (usually colonic), accompanied by a croupous, or true, diphtheritic exudation. Two clinical forms are recognized:

## PRIMARY DIPHThERITIC DYSENTERY.

In mild grades of this rare affection a grayish-yellow, croupous exudate appears upon the inflamed mucosa, with a necrosis of the epithelial layer that is often limited to the top surface of the folds of the colon. In other instances the diphtheritic infiltration involves all the layers of the bowel, which now becomes greatly enlarged, its mucous membrane being converted into a yellowish-brown, thick, elastic mass, sometimes extending along the entire length of the large intestine. The changes may be confined to the circumscribed areas, and thick sloughs may be cast off, leaving behind ulcers of corresponding size and depth. Again, these gross lesions may be limited to certain sections of the large bowel, as the rectum or the flexures of the colon. In protracted cases cylinders of pseudo-membrane of considerable length may become separated and evacuated with the stools.

**Clinical History.**—The affection usually has an acute onset and one characterized by the appearance simultaneously of severe local and general symptoms. There may be an initial chill, and there is fever, which rises rapidly, together with a marked and early appearing prostration and delirium. Severe abdominal pains are complained of, and the discharges are apt to be very numerous, containing shreds and large sloughs, or even tubular pieces, of false membrane. When these elements are present in the stools the latter are of a dark-brown color, emitting a fetid odor and generally containing more or less blood and mucus. Tenesmus may be present, and particularly when the rectum is involved.

The **physical signs** are often prominent. The belly in most instances is greatly distended, and on pressure very tender—signs due to the fact that the lesions are situated chiefly in the large bowel, and not, as a rule, to peritonitis.

The **diagnosis** rests upon the intestinal symptoms and the character of the dejections, associated with a grave general condition suddenly developed in a previously healthy individual.

The **prognosis** is almost wholly unfavorable. Occasionally recovery follows, though more frequently the disease takes on a chronic course.

## SECONDARY DIPHThERITIC DYSENTERY.

Here the lesions are similar in kind to those of the primary form, but in the majority of instances of a less intense grade. Rarely they may be both extensive and severe. This variety is met with as a terminal condition in not a few acute and chronic diseases; among the former it is with great relative frequency seen to develop in pneumonia (Bristowe), and less frequently, though in not rare instances, in typhoid fever, according to my own observation. Among chronic affections, upon which this condition may become engrafted, are nephritis, organic disease of the heart, and pulmonary tuberculosis.

**Clinical History.**—No characteristic symptoms attend upon its invasion. There may be slight diarrhea—two to four liquid stools daily—but it is not often accompanied by tormina and tenesmus, and the discharges rarely contain any noticeable amount of blood or mucus.



Very rarely shreds of pseudo-membrane are passed with the stools. Secondary diphtheritic dysentery often induces fatal asthenia.

The **diagnosis** is in most cases merely conjectural.

**Sequelæ.**—A relapse is most likely to occur, and each attack increases the liability of the patient to subsequent attacks. Moreover, in persons who have recovered from acute dysentery we often observe a disordered digestion and irritability of the bowels. Rarely, chronic nephritis follows dysentery. The most interesting sequel, however, is paralysis, which occurs mainly in the form of paraplegia (S. Weir Mitchell). Stricture of the bowel may be a sequel, but it is surprisingly rare.

**Treatment.**—**Prophylaxis.**—This embraces isolation and a thorough disinfection of the discharges, which contain the specific germ of the disease, as soon as passed. The drinking-water during the epidemic prevalence of dysentery should be thoroughly boiled, and healthy persons should avoid the use of improper food, while unhygienic surroundings (overcrowding, etc.) are to be corrected as far as possible. All sufferers from dysentery must be kept in bed, and should occupy a well-aired apartment.

The **diet** should consist of milk and light animal broths during the period of active intestinal symptoms, and in the amebic form of the disease it is well to allow easily digestible solids, as raw oysters, eggs, well-boiled rice, fowl, fish, etc., in small quantities. During convalescence a return to the usual dietary is gradually to be made.

**Stimulants.**—With the development of asthenia and cardiac failure stimulants must be employed, as in other acute infectious diseases, and alcoholics may be supplemented by the use of strychnin in cases of extreme debility.

**Medicinal Treatment.**—If scybalous masses be passing still, the treatment should be commenced by administering a dose of castor oil or a saline purge, and this may be repeated if necessary. It is well to convert dysentery into diarrhea, thus cleansing the bowel thoroughly, if the case be seen early. In the later stages purgatives are attended with baneful effects.

Ipecacuanha has long been, and still is, regarded as possessing a specific influence in cases of dysentery. Its administration is usually preceded by a dose of opium (laudanum or morphin) which is given when the stomach has been empty for a few hours. Most authors recommend that large doses—gr. xx to 3j (1.29 to 4.0)—should be administered; but it is probable that a small dose—gr.  $\frac{1}{6}$  to  $\frac{1}{4}$  (0.010 to 0.016) every half hour—is quite as effective; and in children the smaller doses are to be preferred and will be found to be quite efficacious. Other remedies should also be employed, and among these opium is particularly beneficial in combination with ipecacuanha or in the form of Dover's powder, which contains both agencies. Three chief symptomatic indications are met by the opium—pain, restlessness, and undue peristalsis—and to obtain the best effects from the opiate it should be administered in the form of morphin hypodermically. In cases in which tenesmus is an unusually distressing feature an opium suppository (gr. ij—0.1296) or laudanum (℥xxx—2.0, by enema) exercises a beneficial effect. Bismuth in full doses is useful (3ss-j—2.0–4.0 every two hours),

and in cases of sporadic dysentery I have frequently found the following formula productive of happy effects:

R. Pulv. ipecac. et opii,	℥ss (2.0);
Bismuthi subnitrat.,	℥ss (16.0);
Salol,	℥ss (2.0).
M. et ft. chart. No. xij.	

Sig. One every hour or two.

Among other intestinal antiseptics recommended in highest terms by some are naphthalin and mercuric chlorid. I am entirely convinced that the vigorous employment of supportive measures (appropriate food, alcoholics if necessary) is of far higher importance than the use of any known medicaments internally, since unfavorable cases tend naturally to asthenia and death, while favorable ones tend as certainly to recovery without energetic medicinal treatment.

Antiseptic irrigation of the bowel would be, if properly carried out, a curative measure, since by this means we may destroy the amebæ, and solutions of numerous antiseptic substances and astringents have been recommended for this purpose. Unfortunately, the bowel is frequently so irritable as to seriously interfere with this mode of medication. If, on this account, large injections cannot be given, small ones should be substituted and the quantity gradually increased. Preliminary to their use we may also employ cocain in the form of a suppository, or a small quantity of a solution of cocain (4 per cent.), or a laudanum enema (℥xxx—2.0, in starch-water), after which a large injection may be tolerated if administered slowly and the flow be interrupted at short intervals. Among the best agents are silver nitrate (gr. ss-j—0.032—0.064—ad ʒj—32.0), tannic acid (1 to 2 per cent.), salicylic acid (1 to 2 per cent.), and mercuric chlorid (1:6000). I have for a number of years been in the habit of employing these astringents and antiseptic solutions alternately, administering each once daily. The tannic-acid and the salicylic-acid solutions are best borne during the more active stages of the disease. The temperature of the water should, at first, range from 100° to 110° F. (37.7°—43.3° C.), and subsequently this may be slightly reduced. The patient during the administration of the enemata should assume the dorsal position or that upon the left side, but in either case with the hips well elevated, so as to aid the flow by gravitation. In amebic dysentery warm injections of quinin (strength 1:1000—1:5000) have been used with good effects by some authors, but with directly contrary effects by others.

*Local means* in the form of hot fomentations, light poultices, and turpentine stupes often afford much comfort. The various complications must be met by appropriate treatment, as under other circumstances.

### CHRONIC DYSENTERY.

This form of the disease almost always succeeds an acute attack. Very rarely is it chronic from the start, and particularly if it be the amebic variety.

**Pathology.**—In most instances the large intestine is still the seat of

ulceration. Some of the ulcers show no signs of healing; in others this process is going on; while in still others it is completed and puckered cicatrices are presented. The ulcers are deeply pigmented, as is the unulcerated mucosa, which often presents a slate-gray or blackish color. The submucous and muscular coats are hypertrophied, as a rule, with occasional narrowing of the lumen of the bowel, and cystic degeneration of the intestinal glands is sometimes observed. It is to be noted that in a certain, though small, percentage of the cases ulceration does not occur, the mucosa everywhere presenting an uneven, puckered aspect, due to deposits of fibrous tissue.

**Symptoms and Diagnosis.**—Many of the most characteristic features of the acute form are either but feebly expressed or altogether wanting. This is particularly true of the tormina and tenesmus. Certain elements found in the stools of the acute type (blood, shreds of pseudo-membrane, and tissue) are also rarely present. True dysenteric symptoms, however, may arise during acute exacerbations and without pain or tenesmus; then from three or four to a dozen or more fluid dejections are passed daily. The latter are often frothy (when starchy articles of food are taken), being composed chiefly of fecal matter and undigested particles of food, with considerable mucus; and in severe forms blood and pus may be constantly present in the discharges. In many cases the stools are semifluid (pultaceous), and rarely they contain scybala; or the rather frequent liquid or semifluid discharges may alternate with constipation. In such instances the lesions are apt to be situated in the lowest portion of the large intestine. The character of the discharges is much influenced by the sort of food taken; thus when a mixed dietary is partaken of, they are thin, more frequent, and contain more undigested masses of food. Gaseous distention of the intestines is often an annoying symptom.

The physical signs are negative, save for slight tenderness along the line of the colon.

**Associated symptoms referable to other organs** are not without value in the diagnosis. The gastric digestion is poor, the appetite generally impaired (though variable), and the tongue is clean, red, and glazed, presenting the appearance of raw beef. There are progressive emaciation and asthenia, which eventually reach an extreme degree. The skin-surface becomes dry, harsh, and cool, the facies grim, the pulse exceedingly feeble, the mental faculties greatly weakened in the advanced stage; and, as in the acute form so in the chronic, death is usually due to asthenia—with this difference, that in the latter the end is reached more slowly. Rarely peritonitis in consequence of perforation of the bowel is the immediate cause of death.

**Differential Diagnosis.**—The disease is discriminated from *chronic diarrhea*, often with great difficulty. In chronic dysentery there is the history of an antecedent acute attack, with the appearance from time to time of exacerbations, at which periods mucus, pus, and often blood are contained in the discharges. The latter are, at the same time, more frequent and apt to be accompanied by more or less abdominal pain and tenesmus, and the presence of these features would serve to eliminate chronic diarrhea. From *tuberculous ulceration* of the intestines it is distinguished by the absence of any history of tuberculosis, family or per-



sonal, and of tuberculous new growths in other portions of the body, particularly the lungs.

The **complications** are the same as in acute dysentery, if we except the greater liability, due to the great and protracted weakness of the patient, to certain serious intervening diseases (chronic nephritis, tuberculosis, pneumonia, etc.). Ulceration of the cornea has frequently been noted.

The **duration** is long, the disease lasting for many months or even several years.

**Treatment.**—This should be directed mainly to the local condition, and should consist in methodic irrigation of the bowel with a view to promoting the healing of the ulcers. Formerly it was sought to accomplish the latter indication by the use of certain remedies internally, as silver nitrate, balsam of copaiba, bismuth subnitrate, etc., but the only preparation which I have found to be useful is the zinc oxid (gr. v—x—0.324–0.648) three times daily. The latter preparation is markedly palliative, sometimes even curative.

*Intestinal irrigation* is to be tried, and should be alternated with various disinfectants and astringent remedies, as advocated in the acute form. Among individual remedies the silver nitrate (gr. ss—ij—0.032–0.129) every second day is doubtless the best. On intervening days antiseptic remedies may be used in solution, such as mercuric chlorid (1:6000) or salicylic acid (1 to 2 per cent.); and of other useful agents I may mention tannic acid, alum, acetate of lead, creolin, and quinin sulphate.

Prior to the use of any of the above-mentioned enemata the bowels should be well flushed with a large injection of tepid water, so as to remove the fecal and other irritating materials. The same details are to be observed in carrying out this mode of treatment as in the acute forms of dysentery. Gallay<sup>1</sup> has related the curative effects of large enemata of a solution of crystallized silver nitrate in distilled water, a scruple to a quart (1.296 per liter), to which 20 or 30 drops of laudanum have been added. Amelioration follows the third or fourth washing, but a course of sixty is recommended to secure permanent relief. I agree with the late Austin Flint that the lower part of the rectum should be examined with the speculum, and appropriate topical applications made if ulcers in this situation be discovered.

The *dietetic* treatment in chronic dysentery is of the utmost importance, and the lightest forms of albuminous foods are to be adhered to strictly, to the exclusion of vegetable substances. Milk is excellent when it can be taken. It is well to examine the stools, and if on microscopic examination curds or numerous fat-globules appear, the amount of milk should be reduced or skim-milk substituted. Other forms of food that are allowable and useful are egg-white, meat-broths or beef-juice, whey, and the like. The patient should wear flannels next the skin, so as to protect against the vicissitudes of weather, and, while open-air exercise is useful, it should be moderate. During inclement weather the patient should remain in-doors. I have known change of climate, with proper regulation of the mode of living, to be productive of rather brilliant results.

<sup>1</sup> "Radical Cure for Chronic Dysentery of Recurrent Type," *British Med. Journal*, No. 1779, p. 276.

Tonics and alcoholic stimulants are sometimes required to assist the appetite, digestion, and systemic strength, and among the most efficacious tonic remedies are iron, strychnin, mineral acids, and arsenic, which may be used in succession.

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## CHOLERA (EPIDEMIC).

(*Asiatic Cholera; Cholera Algida, etc.*)

**Definition.**—Cholera is an acute, infectious, epidemic disease. Its specific cause is the spirillum of Koch, and its most characteristic symptoms are copious watery dejections, painful cramps, collapse, and suppression of the excretions. In some localities it is endemic.

**Historic Note.**—During the Middle Ages cholera made deplorable ravages, chiefly along the belts of the Ganges, and has probably been endemic in India for centuries. Only during the present century, however, has the disease been widely known in Europe and America, and when it has appeared it has always been in the epidemic form. The march of epidemics has been from east to west, and always along the lines of commerce and travel by land or sea, sometimes spreading over the entire globe. While interesting, it would not be profitable to the student to detail here the progress of the various epidemics of cholera in Europe and America. It will suffice to state the years in which the chief of these occurred: in 1831–32, in 1835–36, in 1847–49, being brought by immigrant ships from Europe; in 1852 in Europe (touching our shores in 1854 and prevailing extensively); in 1859 (Europe), in 1866–67 (mild outbreaks in America), in 1869–73 (America in 1873), in 1884 (in Europe), and in 1892–93 (abroad). It is seen that there have been no epidemic visitations in America since 1873, though a few small groups of cases have on several occasions been brought to our shores.

**Pathology.**—The body is generally much emaciated, the features sharp and drawn, and the skin of the dependent parts presents a mottled appearance. A post-mortem rise of temperature often occurs. The tissues are dry, owing to the draining of the liquids of the body, and hence putrefaction is delayed. The kidneys, liver, and heart, as well as other organs in a less degree, show excessive cloudy swelling and often considerable fatty degeneration of the parenchymatous tissues. Rigor mortis comes on directly after death, is persistent, and the muscles often contract so as to cause the body to assume various uncommon positions.

**The Visceral Lesions.**—The chief of these are confined to the intestinal canal, and depend greatly upon the period of the disease at which death occurs. In the early stage the serosa of the small bowel is congested, presenting a roseate hue. The muscularis is relaxed. The mucosa is the seat of catarrh, being deeply injected, swollen, at times edematous, and often coated in the early stage with more or less tough mucus. Shortly the coils of intestine are filled with an almost transparent or slightly turbid liquid ("rice water"), and, occasionally a small amount of clotted blood is seen in the bowel. The solitary follicles and Peyer's patches are at first swollen, and may later, in rare instances, become ulcerated. De-

transudation of the epithelial lining—most probably a post-mortem change—is the rule, and large or small ecchymotic spots are visible in the intestinal mucosa. If the patient has died late in the disease (stage of reaction), patches of false membrane (diphtheritic), sometimes dark-brown in color and fetid, may be found anywhere along the intestinal canal, though chiefly in the large bowel; and this secondary croupous-diphtheritic process may attack other mucous surfaces (bile-ducts, vagina, etc.). The bacilli are observed in the mucous membrane of the intestine and in the dejections.

**The stomach** shows changes similar in character to those found in the intestines. At first the mucosa is congested; then, as the result of transudation, it becomes filled with “rice-water” material. Soon the hyperemic mucosa becomes swollen and ecchymoses appear. At last the organ is empty and collapsed.

**The œsophagus** also exhibits about the same changes, though with an absence of the characteristic transudation.

**The spleen**, contrary to its condition in other infectious diseases, is small as a rule, though if death occur late it may show some degree of enlargement with softening.

**The liver** presents marked passive hyperemia and cloudy swelling, with minute spots of beginning fatty change. Desquamation of the epithelium of the cystic mucosa may occur and lead to a blocking of the bile-ducts.

**The kidneys** show important lesions, being enlarged from passive congestion, especially the cortex, and the capsule being somewhat adherent. They exhibit cloudy swelling and decided coagulation-necrosis. Desquamation of the epithelium in the uriniferous tubules is extensive. Microscopically, the histologic changes are those of acute nephritis in the cases in which death takes place in the advanced stage.

**The bladder-changes** differ in no way from those of other mucous membranes. Its mucosa is congested, ecchymotic, and sometimes the seat of diphtheritic deposit. The ureters and the pelves of the kidneys may also present identical appearances.

**The Circulatory System.**—The pericardium is dry, the parietal layer being covered with an adhesive secretion, while the visceral layer is the seat of more or less ecchymosis. The *heart* is dry and anemic-looking. The left ventricle is contracted, while the right is often distended with blood and soft clots, the latter sometimes extending to the pulmonary artery and the superior and inferior venæ cavæ. Outside of the heart the veins, including the cerebral sinuses, contain most of the blood. The latter is thicker than normal, and its color darker, resembling “the juice of huckleberries;” its specific gravity, albumin, and corpuscles are all increased, while its saline constituents and coagulability are decreased.

**Respiratory Organs.**—The larynx, trachea, and bronchi are hyperemic, and at first covered with tenacious mucus; later they may present ecchymoses and diphtheritic processes.

When death occurs before the stage of reaction the lungs are bloodless and collapsed, and the mouth of the pulmonary artery may be distended with blood. If life is prolonged until the third stage, the lungs may show congestion and edema (particularly at the bases) or pulmonary infarction. The post-mortem of a case in this stage, and especially during convalescence, may exhibit the lesions of broncho- or lobar pneumonia.



The brain and its membranes may be the seat of hyperemia, except when death takes place at a late period, and then the brain-substance may be more or less bloodless and edematous.

**Etiology.**—The causes are (a) *specific* and (b) *predisposing*.

(a) The specific cause is the comma bacillus of Koch, which is found in the intestinal canal of persons ill of cholera. Recent investigations into the bacteriology of the affection show that almost uniformly the cholera spirillum is associated with certain bacteria, most commonly the bacillus coli communis. It has also been shown pretty clearly that true cholera is a *nitrite*-poisoning, the result of the growth of the specific

spirillum. The comma spirillum is not found in any other disease. Its form is that of a slightly curved rod, and its length about half that of the tubercle bacillus, but it is thicker and sometimes has the form of the letter S (Fig. 13). It is to be classed as a spirocheta, and has been grown successfully on media of various sorts and equally successfully inoculated upon inferior animals.

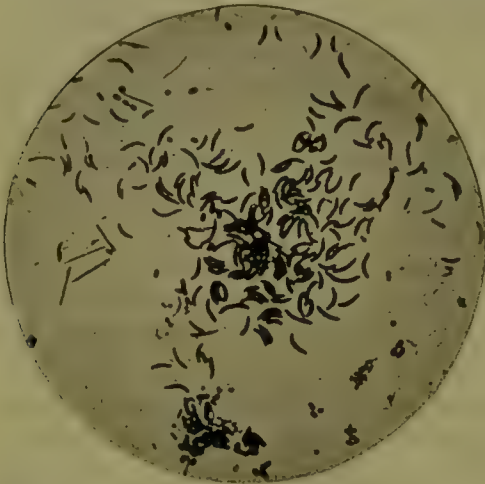


FIG. 13.—Comma bacilli (from the mouth);  
× 1000 (Günther).

The organism is found in a variety of positions—in the intestine, the dejecta (even quite early), and in great profusion in the pathognomonic rice-water stools.

Kemp in his review has shown that the comma bacillus is often absent from the evacuations, and that in these cases the bacterium coli is usually present and sometimes streptococci. He believes, however, that the apparent absence is due to faulty technique. To find it in the vomitus, however, is rare. On the other hand, it may be seen in the stools of well persons during epidemics, displaying virulent properties.

*Cholera spirilla* have been repeatedly found in the outer world, and almost invariably in water. C. Fränkel during the European epidemic of 1892 studied them in flowing water, and in other epidemic outbreaks they have been found in the water used for drinking-purposes.

(b) **Predisposing Causes.**—(1) **Locality.**—Near to the sea-coast cholera is more common than in the inland districts or towns, and the frequency of occurrence lessens with increasing altitude, this fact possibly being due to a gradual decrease in soil humidity and porosity.

(2) **Atmospheric Temperature.**—The spirillum of cholera can only flourish in a warm temperature or in a warm climate; hence the disease is *endemic* in certain tropical and subtropical climates only; and hence we see in temperate latitudes the *epidemic prevalence* of the disease only, and that during the warm season.

(3) **Seasons.**—From what has been stated it may be seen that cholera can have no permanent home except in very warm climates in which all the other essential conditions prevail. For equally obvious reasons it is more common in the warm than in the cold months, most epidemics,

both in Europe and America, having occurred toward the close of summer and in the early autumn.

(4) **Age**, as a rule, has no decided effect. It should be stated, however, that old people are very prone to the affection. **Sex** is without perceptible influence.

(5) **Debilitating Causes**.—Whenever the private conditions correspond to rigid scientific requirements during epidemic outbreaks cholera becomes less prevalent and also less virulent. On the other hand, the deplorable state of municipal sanitation, individual disregard of proper hygienic rules, nervous depression, intemperance, overcrowding, etc. all predispose markedly to the disease.

(6) Mere attacks of **intestinal disorder** due to improper diet, cold, etc. are potent, and are the sole agencies by means of which the disease is disseminated.

**Modes of Infection**.—The spirilla leave the body with the stools, but the most frequent bearer of cholera-poison is the drinking-water. Naturally, the individual susceptibility varies greatly (many persons being even insusceptible), and yet the degree of contamination of the drinking-water and the virulence of epidemics are almost strictly proportionate. As an illustration, Vienna had enjoyed exemption from cholera for nineteen years—a fact attributed to the excellent quality of the drinking-water and to hygienic improvements. In the same city the mortality-rate in the more recent epidemics has been small (7 per 1000) for a like reason. On the other hand, in 1872 there occurred in a single commune (Hamburg), which had a polluted water-supply (the Elbe) and no filtration plant, 17,862 cases, with the enormous death-rate of 42.3 per cent. Biernacki demonstrated the presence of spirilla in the spring-water of a house in which 13 cases of cholera occurred.

The *choleraic poison* may be conveyed with the water used for washing, cooking, and other purposes to other fluids imbibed by man (beer, milk, tea, etc.), and also to food-stuffs taken by him (lettuce, cresses, and other raw vegetables, fruits, meats, bread, butter, etc.). The organisms live and maintain their virulence on these articles of food from four to seven days at least. The infection may reach the esophagus with the water used for washing the mouth or teeth, or that used for washing the utensils, dishes, food-receptacles, etc. Again, the hands, commonly those of laundresses and nurses, may become soiled in the careless handling of bed-linen or garments worn by cholera patients or the stools, and convey the poison to the mouth or lips, to be carried into the stomach along with the drink or food. It is quite possible that flies may transfer the infectious element to food-articles (Simmonds).

Cholera is not contagious from mere contact with those ill of the disease. The disease is not acquired by inhalation (Shakespeare), and, since desiccation rapidly kills the spirillum, there is little probability that the latter is wafted by the wind-currents or is air-borne. Nor is there any clinical evidence to show that the poison may enter the system through the skin-surface. Probably the germs are *swallowed*, and the acid gastric juice may then destroy them if the size of the dose of the poison is not too large, or a sufficient number may pass into the intestinal canal and there manifest pathogenic powers. It is to be borne in mind that after the spirillum reaches the intestine, whether or not an



attack of cholera is the result depends both upon the size of the poisonous dose and upon the personal degree of immunity.

Opposed to the drinking-water theory of this disease is that of Pettenkofer, which contends that the spirilla found in the serous evacuations of cholera patients must enter an appropriate soil and there undergo further development before becoming truly pathogenic. While soils possessing a certain degree of moisture and perviousness and contaminated with organic matter favor the growth and multiplication of the specific organism, these telluric conditions are not essential, as is shown by the virulence of the intestinal discharges when swallowed in ample quantity. Pettenkofer also claims that the fully developed poison rises from the subsoil into the lower atmospheric strata as a miasm, especially at the time of the subsidence of the ground-water level in summer.

*Immunity* is not conferred by a previous attack of cholera.

**Clinical History.**—The incubation period varies from a few hours to five days, and averages about two days. During this prodromal period the patient is either quite well or (during the latter portion) exhibits certain local symptoms. These are occasionally nausea, a feeling of distress in the abdomen, increased peristalsis which may be visible or palpable, slight pain and tenderness, and either a mild or a decided diarrhea. The discharges are feculent, colored, and semifluid, or more rarely quite fluid, and may be quite copious. These symptoms may all be present, though oftener a few, and rarely a single one, is noted; moreover, they are not distinctive unless seen during an epidemic and unless the patient have been exposed to the poison. General symptoms, as a rule, are not present, though rarely prostration may be marked and there may be slight muscular cramps. The so-called premonitory diarrhea may terminate in recovery at the end of from one to three days, but if not it is followed by an attack of true cholera. This has three stages: the **stage of serous diarrhea**, the **algid stage** or collapse, and the **stage of reaction**.

(1) **Stage of Serous Diarrhea.**—The dejections are generally painless, very frequent, odorless, copious, and fluid or watery, and usually present the characteristic “rice-water” appearance. Rarely they are distinctly colored with bile, and in severe cases with blood, and rarely also are they frothy. Suspended in them are numerous small, whitish, mucous flakes; their reaction is neutral or alkaline, and they contain a small percentage of solid constituents made up largely of albumin and sodium chlorid. The microscope brings to view epithelium, mucus, triple phosphates, and numberless micro-organisms, of which latter the only ones characteristic are the comma bacilli (spirilla) of Koch. In *cholera sicca* these serous evacuations are absent. Death comes quickly, and post-mortem examination shows the intestine to be filled with rice-water material, which is probably retained because of almost instant paralysis of the muscular coat of the intestine.

*Gastric symptoms* appear early. Vomiting soon becomes frequent, and at first the vomitus may be bilious; later it is characteristically serous, like the stools, and excessive in amount. Thirst is almost intolerable, anorexia is complete, and the tongue often has a thick coating which early becomes dry. Gastro-intestinal pain is not severe, but a feeling of pressure and of burning in the abdomen is experienced, and occasionally there are griping pains with tenesmus. The physical signs



are few. The belly is usually flat and flaccid, though it may be scaphoid and hard, and in some places palpation detects fluctuation due to the presence of much serous fluid.

*Painful cramps* in the *muscles* form an early and characteristic symptom. They affect the voluntary muscles of the legs and feet (especially the calves), more rarely the arms and hands also. Their duration is only a few minutes, but they recur at brief intervals, and are probably due to the local effect of the circulating toxins.

Owing to the withdrawal of fluid from the lymphatics and blood-vessels the tissues become dry and shrivelled and the blood much thicker. This condition of the blood obviously increases the labor of the heart, which beats rapidly, and there may be at first a distressing palpitation, but soon the heart grows more and more feeble and venous stasis ensues. The pulse is at first rapid, soft, and small; it may then be lost at the wrist. The cardiac impulse may disappear with increasing asthenia—a condition with which the heart-sounds are in direct relation.

The facies and general appearance also indicate loss of fluid. The cutaneous surfaces of the face and extremities grow cool: there is rapid general emaciation, which may become most pronounced, and the skin is wrinkled. The complexion assumes a livid or blue-gray tint, while the lips become quite dark. The extremities are cyanotic (the finger-tips in particular), the orbits are deeply sunken, the cheeks hollow, the features intensely pinched, the voice husky and feeble, and there is utter prostration. The surface-temperature drops below the normal, even to 96° or 95° F. (35.5°–35° C.), while, *per contra*, the internal or rectal temperature rises to 102° F. (38.8° C.) or over. The mind may remain clear until the close, but oftener the patient is apathetic, and in grave cases this condition may deepen into stupor or even actual coma. The reflexes are greatly diminished, and restlessness and jactitation may appear, but rarely.

The *urine* becomes very scanty and is highly concentrated, the standing specimen depositing a heavy sediment. On analysis albumin and casts (chiefly granular) are found. In the serious forms the kidneys fail to eliminate the urea, and there is finally complete anuria, which may last for a couple of days or until life is terminated.

(2) *Stage of Algidity or Collapse*.—The symptoms which characterize this grave condition are the same as those noted under the latter part of the first stage, only intensified. Asthenia is extreme; the pulse is missing and the heart beats faintly; the voice is lost; respirations are perceptibly shallow; lividity is intense; the surface ice-cold; and there is usually stupor or even coma. The excessive serous discharges have given place to mere dribblings from the now relaxed anus. During this stage, which may last a few or many hours, the faint glimmerings of the vital spark are often extinguished.

(3) *Stage of Reaction*.—This sets in promptly, and leads as promptly to complete recovery after a mere “premonitory diarrhea;” and when reaction follows the first stage directly the case may pursue a favorable course, with return to accustomed health by the end of a week or ten days. The first urine passed is usually albuminous and contains tube-casts and sometimes blood-cells. Relapses into the stage of collapse may occur and be repeated; in many instances, however, this stage is both protracted and

dangerous. It is aptly termed *cholera typhoid*, since a genuine typhoid state of the system with more or less fever develops. The skin may present so-called choleraic eruptions (macular, roseolar, erythema, purpura, etc.). Recovery may now take place, or a great diversity of local secondary inflammations may supervene (*vide* Complications).

*Acute nephritis*, which may or may not be an essential part of the process, may arise in this stage and lead either slowly or directly to uremic poisoning, as shown by the projection upon the clinical picture of grave nervous phenomena—headache, vomiting, delirium or coma, and convulsions. A fatal result may be looked for.

**Complications.**—In this place are to be enumerated the conditions due to secondary infection, including (most commonly) septic and pyemic processes. Diphtheritic inflammations affecting most mucous surfaces, but especially the throat, colon, and the external genitals, are among the more common. Bronchitis, pneumonia, and pleurisy may arise, and erysipelas and parotitis are not rare. During convalescence digestive disorders may show themselves, and indiscretions in diet may precipitate a relapse.

**Clinical Types.**—(a) “Premonitory Diarrhea.”—This type has been outlined in the foregoing discussion, and will not need further description.

(b) “Cholérine,” in which the symptoms are similar to those of cholera nostras. Many of the symptoms characteristic of true cholera are also present, particularly the cramps and prostration, cold extremities, and scanty albuminous urine. The stools, however, are not, as a rule, typical of the disease, but are feculent in character, as in ordinary cholera morbus. The duration is from seven to ten days, subject to relapses.

(c) The more typical forms—both moderate and severe—have been described under the Clinical History (*supra*).

(d) The Foudroyant or Asphyxic Form.—This may kill instantly; more frequently the patient lives for a few hours, with or without vomiting and purging. *Cholera sicca* should be classed with this type. The virulence of the cholera-poison explains the intensity of the symptoms.

**Differential Diagnosis.**—This is difficult in the absence of an epidemic unless bacteriologic and microscopic tests be made, and yet these alone differentiate a sporadic case. The disease most commonly mistaken for cholera (especially cholérine) is *cholera morbus*, and the following points pertaining to the latter disease will eliminate it: 1. No connection with a previous case, but a frequent history of dietetic imprudence. 2. Absence of “rice-water” stools, which remain turbid with feces or covered with bile or blood. 3. Presence of colicky pains, but absence of painful tonic cramps of legs and feet. 4. Absence of cyanosis and collapse, as a rule, and of urinary suppression. 5. No cholera spirilla in the stools.

*Arsenic-poisoning* and other forms of *gastro-enteritis* must be discriminated by the history, the character of the stools, the absence of violent muscle-cramps and of the effects of great loss of fluid (cyanosis, shrunken body, profound collapse, etc.). Chemical tests are not to be neglected if the history points to any form of corrosive poisoning.

**Prognosis.**—This is dependent mainly upon the type. Thus “cholérine” is very rarely fatal, while, on the other hand, the asphyxic form is almost as rarely survived. It is impossible to state the average mortality,



since it varies with each epidemic, but it has been found to range from 20 to 80 per cent. Many deaths occur during the latter part of the first day or during the algid period; still more during the stage of reaction, the dangers of the latter period being as follows: asthenia, cholera nephritis with uremia, and the various complications (*vide supra*). The personal circumstances which render an attack grave are old age, alcoholism, previous ill-health, and debility. On the other hand, the death-rate may readily be lowered by prompt and judicious treatment.

**Treatment.**—**Prophylaxis.**—Prevention is of greater importance than cure, and is easily accomplished as compared with the eradication of the disease. It has been owing in great measure to the efficient quarantine system of the United States that cholera has not gained a foothold on our shores since 1873.

*Individual Prophylaxis.*—In the first place, those nursing the sick can prevent the spread of cholera by prompt and thorough disinfection of the vomitus and stools, as well as of the receptacles containing them and anything that may be soiled by them. The dejecta may be disinfected by pouring upon and mixing with them an equal part of a 5 per cent. solution of carbolic acid or an equal volume of a freshly prepared solution of chlorid of lime. The discharges thus treated must be covered and allowed to stand from fifteen minutes to half an hour, and then emptied into a pit in the earth containing quicklime, with which they should also be covered. It is of the utmost importance to guard against a pollution of the water-supply by these pits. Soiled clothing, linen, etc. should be promptly disinfected, and bedding had better be burned; none but the attendants should be permitted to enter the sick-room. The dishes used should be disinfected immediately after use or before leaving the sick-chamber. Shakespeare further recommends that the remains of the patient's meals should be disinfected and destroyed. After handling the patient or anything that he has soiled the attendants should first disinfect and then carefully wash their hands, these ablutions being performed invariably before eating. After vomiting and after an evacuation of the bowels the mouth and the parts around the anus should be wiped with a cloth wet with a solution (1:2000) of mercuric chlorid. If convalescence supervene, the patient should be kept isolated for a week and the stools should be disinfected during that time.

*Persons exposed* should use boiled milk and water only. Certain forms of food must be avoided, especially salads and unripe fruits; also alcoholic stimulants. All uncooked food may be pernicious. Such persons should lead regular lives, avoiding fatigue, excesses, etc., and intestinal disturbance must be met speedily by the use of antiseptics, opiates, and astringents. In India, Haffkine<sup>1</sup> has used a protective virus with encouraging results. Thus, "of 1735 persons not inoculated in a certain section, 174 took the disease and 113 died, whereas of 500 inoculated but 21 were affected and 19 died." He has made, altogether, 70,000 injections in 40,000 patients without a single accident, and claims that the results have been entirely favorable. Behring and Ransom have also succeeded in obtaining an antitoxic serum. Klein concludes against Haffkine's anticholera inoculations, showing that there is no certainty as to the protection against the specific poison in the intestines, even

<sup>1</sup> *Münch. med. Woch.*, Jan. 29, 1895.



though there may be protection against the effect of intracellular poison. Klemperer has produced immunity by using a toxin.

**Treatment of the Attack.**—(a) **Premonitory Diarrhea.**—When the prodromal period exists it must be quickly combated, and if this were attended to appropriately few cases of cholera would follow. In the instances which are not preceded by premonitory diarrhea opportunity to prevent the attacks does not present itself. In this stage a double indication is presented—"to restrain the development of the bacilli in the intestine and to neutralize the cholera-poison." To meet this Cantani proposes tannic acid by irrigation (enteroclysis). He injects into the intestine  $\frac{1}{2}$  to  $2\frac{1}{2}$  quarts (liters) of water, or infusion of chamomile containing  $\text{ziss}$  to  $\text{zv}$  (6.0 to 20.0) of tannic acid, gtt. xx to xxx (1.20) of laudanum, and  $\text{zv-xij}$  (20.0–50.0) of gum arabic. The temperature of the liquid should be  $80^{\circ}$ – $104^{\circ}$  F. ( $26.6^{\circ}$ – $40^{\circ}$  C), in order not to chill the patient. Injections should be repeated four times a day, and in grave cases after each evacuation.<sup>1</sup> To this should be added a regulated liquid diet, with rest and recumbency. For the same purpose acetate of lead and opium, or large doses of bismuth with or without Dover's powder, have been much employed with good results.

(b) **Stage of Serous Diarrhea.**—The chief indication is to restore to the blood the watery elements withdrawn by the diarrhea. Not a moment is to be wasted. Opium, and preferably the salts of morphin, should be administered hypodermically, the dose not being small, but gr.  $\frac{1}{4}$  to  $\frac{1}{3}$  (0.0162–0.0216) to be repeated at intervals of about eight hours. To opium given *per oram* or in the usual way there is a serious objection—namely, its slowness of action. Cantani advocates the injection of an artificial serum (*hypodermoclysis*) containing 1 dram (4.0) of sodium chlorid and gr. xlvj (3.0) of sodium carbonate per quart (liter) of sterilized water warmed up to  $104^{\circ}$  F. ( $40^{\circ}$  C.) into the subcutaneous connective tissue. This solution may be introduced through the cannula of an ordinary aspirator, the fluid flowing by gentle pressure. Shakespeare recommends for hypodermoclysis a fountain syringe with a long flexible tube furnished with a cock; with another shorter tube, one end attached to the cock, the other having a needle-pointed cannula, a little longer, stronger, and with a somewhat wider caliber than the ordinary hypodermic needle (Fig. 14). The tube and cannula are first perfectly filled with a fluid, and then the cannula is inserted well in between the skin and deep fascia of the flanks, buttocks, or interscapular region. The fluid should be made to flow slowly, allowing fifteen to twenty minutes for the introduction of one quart. This is preferred to intravenous injection, in which the liquid is diffused slowly. The indications presented by the premonitory stage must be met as above stated.

The *vomiting* is to be relieved by bits of ice, small amounts of brandy and water at brief intervals, cocain, or by *lavage*. In this stage remedies by the mouth should be avoided, since they aggravate the gastric disturbance. Heat should be applied externally with a view to assisting the peripheral circulation as well as the reaction, and, on the other hand, to obviate collapse. Warm baths have been recommended for this purpose. Stimulants must be used to fulfil the same indications. They are of superior value even to the above-mentioned measures, and are to be

<sup>1</sup> *Annual of the Universal Medical Sciences*, 1893.

given hypodermically, and either brandy, ammonia, or strychnin may be employed in large doses.

(c) **Stage of Algidity.**—If this develop, the case is desperate. In this stage the following measures and procedures, which have been detailed in the treatment of the preceding stage, are to be persevered with:

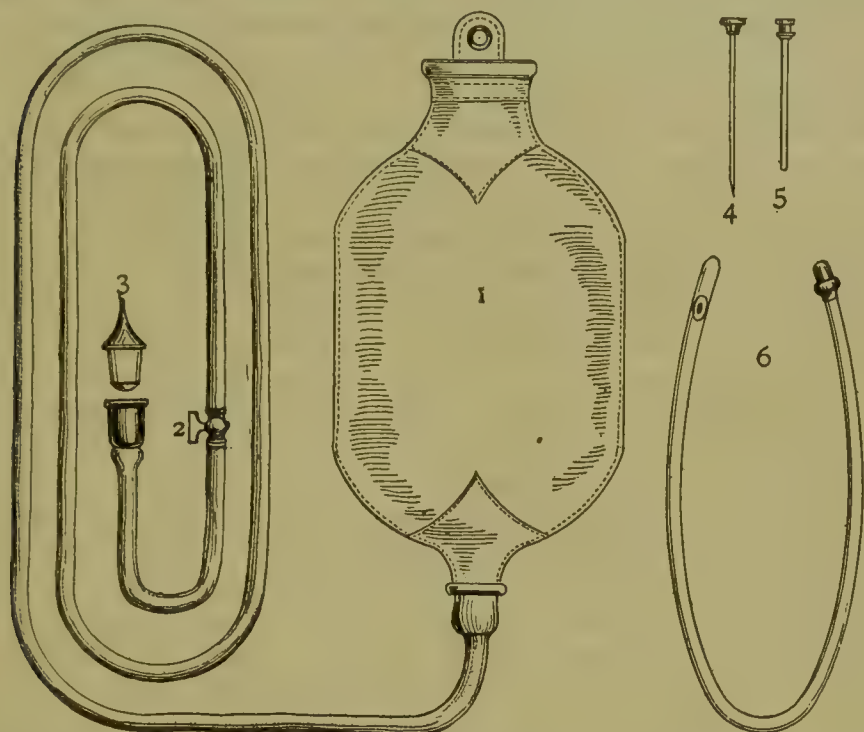


FIG. 14.—1, fountain syringe; 2, cock; 3, attachment for cannula; 4, needle; 5, cannula; 6, soft-rubber rectal tube, with two lateral openings, one a half inch from the end (not visible), the other two inches from the end. The latter is to be introduced by a combined rotatory and pushing motion to the depth of ten inches in enteroclysis, and the fluid then allowed to enter the colon slowly.

enteroclysis and hypodermoclysis, hypodermic stimulation, and the external application of heat. Additionally, intravenous injections of fluids have been strongly urged by its champions. For this purpose the following standard of saline fluid may be chosen: sodium bicarbonate 1 part, sodium chlorid 6 parts, boiled water 1000 parts. The temperature of the fluid when injected varies according to circumstances from  $100\frac{2}{5}^{\circ}$  to  $104^{\circ}$  F. ( $38^{\circ}$ – $40^{\circ}$  C.), more frequently the latter (Shakespeare). The quantity demanded may be 1 or 2 quarts (liters), and the injection may need to be repeated in from one to three or four hours. Despite the physician's best efforts, patients in this period usually succumb.

**Treatment in the Stage of Reaction.**—During this stage the tannic acid may be replaced by a solution of salt in water (10 or 15 per cent.) for enteroclysis (Cantani), and it may be well to continue hypodermoclysis in some instances. Further than this, the treatment is essentially symptomatic. Food of the blandest sort and in small quantities must be allowed at frequent intervals if we would avoid enteritis and other unfavorable complications. Tonic remedies should be given cautiously, and rest and careful nursing insisted upon. Complications must be met in accordance with general principles.

## YELLOW FEVER.

(*Febris flava*; *Gelbfieber*, Ger.)

**Definition.**—Yellow fever is an acute, highly infectious (but non-contagious) endemic and epidemic disease. It is characterized by a sharp period of invasion, followed by a period of remission, and the latter in turn by a relapse and certain symptoms peculiar to the affection (black vomit, jaundice, suppression of urine).

**Historic Note.**—Yellow fever is endemic only within certain geographic limits, where it also prevails epidemically when the conditions are favorable. According to general belief, it first appeared in 1647 in the Barbadoes (West Indies). Subsequently, it was conveyed along the channels of commerce until it became widely disseminated, and chiefly in sea-port towns. In 1699 an English vessel carrying slaves transported the disease to Mexico from the Atlantic coast of Africa. Guit  ras classified the areas of infection thus: (1) The *focal zone*, in which the disease is never absent, including Havana, Vera Cruz, Rio, and other Spanish-American ports. (2) *Perifocal zones*, or regions of periodic epidemics, including the ports of the tropical Atlantic coast in America and Africa. (3) The *zone of accidental epidemics*, between the parallels of 45   N. and 35   S. latitude. Yellow fever was brought to the United States (Boston) in 1693, and since then has invaded in epidemic form numerous sea-coast cities, being carried thence to a number of inland towns. The belief that the disease never originates outside of certain territorial limits was advanced for the first time by the College of Physicians of Philadelphia (1797), and the efficacy of rigid quarantine regulations in preventing conveyance of the poison by vessels having yellow-fever cases on board was pointed out by the same organization.

**Pathology.**—The *skin* is jaundiced (hepatogenous) and often large or small ecchymotic spots are observed, but neither the internal viscera nor the blood shows characteristic lesions in cases of average intensity. In severe forms congestion, hemorrhage, and degeneration are the changes noted, especially in the liver and the gastro-intestinal mucous membranes.

After death the liver is anemic, as a rule, but in the early stages of the disease it is markedly hyperemic. Its color varies, ranging from pale yellow to an orange hue, and punctiform extravasations cause mottling of the surface. Its size varies little from the normal. Parenchymatous degeneration of the hepatic tissue is common, though in places it may be entirely normal. The liver-cells are swollen, containing fat and granular matter with indistinctness or absence of nuclei.

The *gastro-intestinal mucosa* is the seat of numerous minute hemorrhages, similar spots of extravasation being found on the various serous membranes of the body (meninges, pericardium, pleura, etc.). Hemorrhagic infarctions may be found in the various internal viscera. The lesions of acute catarrh are seen in pronounced form in the gastric mucosa, which may also present erosions. The black-vomit material is found in the stomach, and less frequently also in the smaller intestines, which present the evidences of acute catarrh of their mucous walls.

The *spleen* is dark and friable, but is not enlarged. The *kidneys* show the lesions of parenchymatous nephritis, the microscope revealing



cloudy swelling of the epithelium of the tubules with fatty degeneration, and the tubules themselves being occupied by casts, chiefly granular. The heart-muscle looks pale, and may be the seat of granular and fatty degeneration. The *brain* and its *meninges* are hyperemic, and degenerative changes have been described in the sympathetic ganglia (Schmidt).

The *blood* is dark, and many of the red corpuscles, having disorganized, set free hemoglobin, as in malaria. Certain significant lesions of a general character—such as a fatty degeneration of the walls of the small blood-vessels and the capillaries—have been noted by competent observers, and these, by allowing filtration of the blood-serum through the vessel-walls, may account in great measure for the concentration of the blood.

**Etiology.**—The fact that yellow fever occurs chiefly in epidemics of varying extent must be borne in remembrance, and when the first cases appear in a fresh outbreak we may feel certain that the disease has been transported from some distant point. Its *bacteriology* is as yet an unsettled problem.

Among predisposing causes *season* heads the list. The disease prevails chiefly in summer, being completely arrested by one, or at most two, severe frosts. The affection is to a far greater degree under the influence of the temperature than of any other meteorologic element, requiring, as it does, at least 72° F. (22.2° C.). Long-continued elevated temperature therefore favors a spread of the disease, and atmospheric humidity also favors its propagation. The poison is more virulent at night than in the daytime; and unhygienic conditions, as overcrowding, filthiness, ill-ventilation, etc., are important disposing factors. *Age* and *race* have some degree of influence, children being more liable than adults, males than females, and whites than blacks. Other factors worthy of special mention are—*intemperance, physical exhaustion, sexual excesses, fear, depressing emotions*, etc. One attack usually bestows permanent immunity, and natives of an infected district are far less liable to the disease than newcomers. The poison is not given off by the severe form of yellow fever in the mature or active form, but only becomes so after it has undergone further development in a favorable soil. It may, however, be carried to short distances by *currents of air*, and may be transferred over any distance by *fomites*—clothing, baggage, letters, and packing-cases being the most frequent carriers. The specific poison manifests great tenacity of life. In a large city the infected district may be small, and, although it tends to extend itself, the process is a slow one. The march of an epidemic may be interrupted or even completely arrested by apparently trivial agencies—*e. g.* watercourses, rows or clumps of shrubbery or underbrush, high fences or walls, and so on.

**Clinical History.**—**Incubation Stage.**—This varies greatly, ranging from one day to two or even three weeks. During the incubation certain general symptoms may appear, such as languor, headache, anorexia, etc., lasting several days.

**Invasion Stage.**—The onset is abrupt, an initial chill usually occurring, but it is very seldom severe or prolonged, a reactionary fever following promptly and the temperature rising to 103°, 104°, or even 105° F. (40.5 C.). The temperature is apt to be highest at the beginning, and then falls gradually, hyperpyrexia being rare. The chill and fever are

accompanied by headache and pains in the loins and legs, often of great severity, and a little later restlessness, mental confusion, and a delirium that is sometimes violent in character may develop. In the majority of instances, however, the mind remains clear. The pulse is accelerated, but not in proportion to the height of the temperature, and is full and strong at the start, diminishing in strength and frequency soon after. The face is flushed and in severe forms quite dusky. The eyes are suffused and intolerant of light. The tongue may or may not be coated, and nausea and vomiting may occur, the latter being one of the most characteristic symptoms of the disease. Associated with these symptoms there are epigastric oppression and burning sensations, with decided tenderness. The vomitus may be blood-streaked or contain chocolate-colored particles, and occasionally unaltered blood is vomited. Constipation is usually present, the stools showing a deficiency of bile. The urine is diminished in amount, dark-colored, and often contains a slight amount of albumin: this *early transient albuminuria* is a very characteristic symptom. The initial stage may last from six or eight hours to two or three days, or even longer, and is longer in the milder forms of the disease. With the termination of this stage the fever remits and the other symptoms disappear with surprising rapidity, the pulse becoming remarkably slow.

**Stage of Remission.**—From this moment convalescence may begin and proceed to full recovery without interruption, the happy event being often marked by critical discharges. In most instances, however, the patient presents certain symptoms and signs of ill-health during this period (more or less prostration, epigastric distress with tenderness, mental dulness or even stupor, and a yellowish tint of skin and urine), which lasts from a few to twenty-four hours, when another stage with its more striking symptoms supervenes.

**Stage of Secondary Fever or Collapse.**—The patient becomes extremely weak, presenting the signs of profound collapse. The surface of the body is cool (extremities often positively cold), the skin in nearly all instances assuming a yellow or bronzed tinge, from which the disease receives its name. The pulse is rapid and compressible, and soon vomiting becomes very distressing. Hemorrhage into the stomach generally occurs, the blood being acted upon by the gastric secretions, and producing the material which is expelled as the characteristic “black vomit.” Occasionally unaltered blood may be vomited; the stools also may be tarry. In the worst cases hemorrhages from other mucous surfaces are common (epistaxis, hematuria, metrorrhagia, etc.), and cutaneous hemorrhages also now occur. In this stage the tongue becomes dry, brown, or even black; less frequently it is smooth, red, and fissured, and sordes may often be observed on the teeth and lips.

In most cases the urine is deficient, containing albumin and casts, and in rare instances there is complete anuria. The latter may precede the development of grave nervous symptoms, as convulsions, or even coma, which may be uremic.

In some instances the temperature rises during this period (secondary fever), and in favorable cases terminates by lysis, or it may assume the typhoid form and result fatally. In all cases that pursue a favorable course convalescence is slow and gradual, and may be uninterrupted by



relapses, but this is an unusual course of affairs. The duration of the entire attack (composed of three stages) is variable, though as a rule it covers about one week.

**Clinical Varieties.**—Many different varieties have been described, each characterized by one or more prominent features, but none seem more justifiable than Finlay's<sup>1</sup> classification, in which he distinguishes three forms: (1) the *acclimation fever*, or *non-albuminuric yellow fever*; (2) the *plain albuminuric yellow fever*; (3) the *melano-albuminuric yellow fever*, characterized by the presence of blood or "black vomit" in the stomach or intestines.

**Diagnosis.**—The symptoms that justify a diagnosis in the initial stage, provided an epidemic be prevailing, are the sudden onset, headache, severe lumbar pains, peculiar facies, nausea, and vomiting of biliary matter. In the early stage intense capillary congestion of the surface of the body is diagnostic and indicative of a severe form of the disease. In the third stage the coexistence of jaundice, the black vomit, and suppression of urine, with evidences of collapse, makes the diagnosis easy. The first cases that appear in a locality hitherto free from the disease are often diagnosticated with difficulty, especially if they present anomalous characters. In such, a certain diagnosis is possible only by exclusion.

*Pernicious malarial fever* has not the deep jaundice, the slow pulse, the peculiar temperature-curve, the intense capillary congestion of the surface of the body, the black vomit, the early albuminuria, and the clear mind—all symptoms that mark yellow fever. On the other hand, the organism of Laveran is pathognomonic of pernicious malarial fever, as is the effect of quinin upon the disease. Kemp has made a microscopic, spectroscopic, and chemical study of the black vomit of yellow and malarial fevers, and found that the pigment in each case was derived from the blood, which had been acted upon by the juices of the stomach. The vomitus in malarial fever, however, contains in addition considerable quantities of bile-pigments and bile-salts, which are wanting in that of yellow fever. Further, in the latter the vomited matter is much more highly acid than that of malarial fever.

**Prognosis.**—Different epidemics show widely different death-rates, and the most potent factor is the particular type of the disease in individual epidemics. Some have been characterized chiefly by the lighter forms of the affection, and in such the death-rate has been as low as 1 per cent. In other epidemics the type of the affection has been so virulent as to make the mortality list run extremely high, even to 100 per cent. In general terms, mild epidemics give a mortality of 5 to 10 per cent., and severer forms one of 30 to 50 per cent. The death-rate is lower in private than in hospital practice.

Among the gravest symptoms *intense capillary congestion*, coming on during the first stage, deserves special emphasis. Equally serious, in most cases in which they occur, are *suppression of urine*, *intense jaundice*, and *uremic toxemia*. The black vomit is not as fatal a sign as the symptoms previously mentioned.

It has been noted that a larger number of men, proportionately, than women and children succumb to the disease, and that it is less fatal among negroes than among whites.

<sup>1</sup> *Edinburgh Medical Journal*, Edinburgh.



**Treatment.**—The measures that are employed in yellow fever may be considered under three main heads: (1) Prophylaxis; (2) general management; and (3) medicinal measures.

(1) **Prophylaxis.**—The patient must be quarantined, and if the area in a city that is infected be definitely known, it should be shunned by well persons, and particularly if the latter are not acclimated. Persons living in infected localities who have not been immunized by a previous attack had better go elsewhere if such a course be practicable. Every available means to prevent a dissemination of the poison by fomites must be enforced, and most important is the thorough disinfection of all personal belongings, bed- and body-linen, mattresses, clothing, etc. The room occupied by the patient must also receive proper attention.

Dr. Domingo Freire has introduced and advocated protective inoculation with diluted virus, and Ashmead<sup>1</sup> recommends immunization after Murray's method, as follows: (1) Instead of carrying the patient to the infected region, take the infection to the patient; inoculate with the blood-serum of a partially-immune subject (a negro), and inoculate a second time with perfectly-immune blood-serum of a white subject who has had yellow fever. (2) Expose infected blood-serum to frost before inoculating, and follow this at once with a second inoculation of immune blood-serum. Frost always modifies the virus.

(2) **General Management.**—The sufferer from yellow fever must be put to bed at once, and an abundance of fresh air (without exposure to strong drafts) must be supplied. The medicaments and the nourishment are to be administered through a tube or spout-cup, so as to obviate raising the patient's head. Body- and bed-linen should be kept scrupulously clean, being changed frequently, and the patient must not be allowed to leave his bed on any account. The *diet* should be of the lightest sort and entirely liquid, beginning with peptonized milk, koumiss, or light broths, and in small quantities.

(3) **Medicinal Measures.**—At the outset it is well to gently stimulate the various excretory organs, and mild laxative diaphoretics and diuretics answer this purpose. Hydrotherapy may be employed to maintain the nervous tonicity and reduce the temperature, but when the spontaneous fall of temperature sets in this method must be promptly discontinued. During the first stage the neuralgic pains, which attack principally the head, loins, and nerve-trunks, are to be relieved by morphin given hypodermically; and for the same symptom Bemiss highly recommends quinin by the rectum (gr. xx—1.296). Intestinal antiseptics may also be used throughout the attack (salol, betanaphtol, etc.).

During the stage of *remission* the powers of the system are to be fully maintained by a suitable dietary and by tonics and stimulants if required.

In the last stage, which generally supervenes, supportive measures must not be forgotten, everything that gives promise of aiding the vital powers being brought into prompt requisition. *Rectal nutrient enemata* should be employed if marked gastric irritability prohibits feeding by the mouth. *Stimulants* are demanded, and these should also be administered per rectum if not retained by the stomach, or they may in some measure be administered hypodermically. The stomach is, as a rule, more tolerant of iced champagne than of other forms of stimulants.

<sup>1</sup> *Medical Record*, New York.

If irritability of the stomach be present, ice and hydrocyanic acid may be tried. Sodium bicarbonate (gr. x to xx—0.648 to 1.296) in Vichy, Apollinaris, or Seltzer water is a most useful remedy, and Sternberg has used it in combination with mercuric chlorid with success in the following formula:

Ry. Sodii bicarb.,	ʒiv (16.0);
Hydrarg. bichlorid.,	gr. ss. (0.032);
Aquæ puræ,	Oj (480).—M.

Sig. For a severe case two teaspoonfuls every hour, day and night; for a mild case, every hour by day and every two hours by night; administer always ice-cold.

Perhaps the chief indication for the use of sodium bicarbonate is the extreme acidity of the various secretions, especially the gastric and renal. Sternberg contends that by fulfilling this indication we prevent in great measure the occurrence of acute nephritis and suppression of the urine. Hemorrhages and other symptoms must be treated by the usual means. If the stage of convalescence be reached happily, tonics (especially quinin) are to be administered, and the customary diet can gradually be resumed.

## CEREBRO-SPINAL MENINGITIS.

(*Spotted Fever; Cerebro-spinal Fever.*)

**Definition.**—An infectious disease, caused most probably by the *Micrococcus lanceolatus*. It is characterized anatomically by inflammation of the meninges of the brain and spinal cord, and clinically by an irregular course, a moderate febrile movement with somewhat characteristic and profound nervous symptoms (excruciating headache, pain in the back and upper part of the spine, contraction of the muscles of the nucha, hyperesthesia, delirium, and oftentimes coma). The disease may occur sporadically or in epidemics, or may even assume pandemic proportions.

**Historic Note.**—Cerebro-spinal meningitis was first recognized and described as late as the beginning of the present century (1805) by Vieusseux of Geneva. During the next decade numerous limited epidemics were observed both in Europe and the United States, and subsequently recurring epidemic and pandemic visitations were noted, though at comparatively long and variable intervals of time. In nearly all the large cities in this country it may be said to have become endemic, and in Philadelphia since 1863; yet the affection is, without doubt, becoming less and less prevalent.

**Pathology.**—The cases that prove speedily fatal do not present gross characteristic changes, but by the aid of the microscope leukocytes are discovered immediately around the cerebral vessels, and round cells in the cortex of the brain. In some cases the characteristic evidences of



encephalitis are already noticeable. On the other hand, the cases in which death occurs after the disease has been fully developed show the lesions of suppurative inflammation of the meninges of the brain. The arteries, veins, and sinuses are much engorged; the ventricles are distended with liquid, but the pia mater is principally affected, its vessels being greatly enlarged, and a more or less copious sero-fibrinous or sero-purulent exudate occurring into the meshes of its network. The longer the duration of the case the more purulent is the exudation. The ventricles of the brain are filled with a similar exudation, and red blood-globules may be present at an advanced stage. The color of the exudate is at first almost clear (being composed of serum); it then changes to a milky turbidity, to a pale yellow, and at last, when it becomes thick, takes on a greenish-yellow color ("leek-green"). The subarachnoid space may be occupied by a uniform layer composed of fibrin and pus, which exhibits the greatest thickness along the longitudinal fissure.

The brain-matter is congested, and sometimes softened in spots, and on section the gray matter may present punctate extravasations. When resolution occurs recovery may be complete, but more frequently the pia mater remains thickened, and here and there are imbedded in its tissue flattened cheesy masses.

The exudation may follow the auditory and optic nerves along their lymph-sheaths, and pus has been found in the internal ear as well as in the chambers of the eye. When the cases tend to become chronic the membranes are thick and adherent, while the cortex shows areas of softening or atrophy.

The membranes of the spinal cord manifest lesions identical with those of the brain. They are vascular engorgements, followed by sero-fibrinous, and later still by sero-purulent, exudation beneath the arachnoid. The changes are more marked on the posterior than the anterior surface of the cord, and the exudate increases in amount in passing from above downward, in severe cases sometimes assuming the form of a sheath which completely surrounds the cord throughout its entire length. The pia mater is congested, and may be thickened, shaggy, and in places adherent to the cord, of which the gray matter may be the seat of serous infiltration, and rarely of softening.

The lungs may exhibit the changes peculiar to bronchitis or pneumonia. In the heart endocarditis may be noted, though rarely, and both the pleura and the pericardium may show inflammatory lesions and contain a serous or sero-purulent exudation. I have noted one malignant case in which hemorrhages into the serous membranes and into the skin had taken place. The spleen may be moderately enlarged, the increase in size and the degree of fever being proportional, and the liver is hyperemic. The kidneys are congested, and bacterial forms have been found associated in the latter with the lesions of acute nephritis and hemorrhage—conditions of which they were most probably the cause.

**Etiology.**—**Bacteriology.**—It is probable that the *Micrococcus lanceolatus* is the specific cause of cerebro-spinal meningitis. Flexner and Barker found the micrococcus uniformly present in all cases in an epidemic which occurred at Lonacoming, a mining town in Maryland.<sup>1</sup> Other bacteria are, however, constantly found associated (*streptococcus pyogenes*,

<sup>1</sup> *Annual of the Universal Medical Sciences*, 1895, vol. ii. A-65.



staphylococcus aureus, etc.), and there is little doubt that these, sometimes give special characters to the clinical bacteria.

**Predisposing Causes.**—(1) **Age.**—Most cases occur among children and young adults, though no age enjoys perfect immunity. Occasionally epidemics have affected adults chiefly.

(2) **Climate.**—The disease is unknown in tropical climates, but has occurred in all parts of the temperate zone, and is most prevalent in the more northerly portions of the latter.

(3) **Season** is not an important factor, though the disease prevails largely in cold weather.

(4) **Unhygienic Influences.**—Those who live under unfavorable sanitary influences are especially liable, and hence the disease often appears in ill-ventilated and overcrowded habitations—among the poorer classes, among soldiers crowded together in barracks, and among prisoners. For like reasons excessive physical or mental exertion or bodily fatigue, as after prolonged marching, may heighten the susceptibility to the disease. In certain epidemics the disease has raged exclusively in villages that afforded the least hygienic conditions.

Cerebro-spinal meningitis is not unfrequently associated with other epidemic affections, especially scarlet fever and measles.

**Modes of Conveyance.**—Precisely how the contagion is transferred from an infected person to a healthy one is not known, though the disease is probably not contagious. There is considerable evidence to show that the poison may be conveyed by *fomites*, though even this seems to be limited to the cases furnishing intensely virulent poison. As to the manner in which infection occurs or the virus gains entrance to the system our knowledge is very imperfect.

**Clinical History.**—The period of *incubation* must be brief, though its duration can only be approximated in the present state of our knowledge. The prodromal symptoms exhibit considerable variety in different epidemics, and may even be absent when the invasion is sudden, a patient in vigorous health often being stricken down as though by a blow. In some of these rapidly fatal cases there is a short prodromal period, during which the patient complains of lassitude, headache, rachialgia, muscle- and joint-pains, and sometimes nausea and vomiting. In ordinary forms the premonitory symptoms may last from a few hours to a week or more, and the patient's complaint may be limited to cervical and occipital pains lasting a day or two; then, without any initial chill, the *invasion period* supervenes. In milder, and usually in sporadic, cases the symptoms consist chiefly of languor and debility, headache, pain in the back and limbs, vertigo, vomiting, and sometimes diarrhea.

Most cases begin abruptly, and with few exceptions the hour of onset is between noon and midnight. The symptoms which are often most distinctive and violent are chill (often severe), fever of a moderate grade, a full and somewhat accelerated pulse, raging headache, and vomiting. In children the ushering-in symptom may be a convulsion. These symptoms are followed in the course of a few hours by pain in the back and cervical portion of the spine—an early and characteristic symptom. Attempts at flexion or rotation of the head increase the pain in the neck, and in like manner movements of the body augment the spinal pains. Later, the muscles in the cervical region contract, at the same time becoming rigid,

and produce the condition of opisthotonos. The patient may be unable to swallow on account of the excruciating pain which the act is apt to excite.

The *temperature* is but moderately elevated. In a certain percentage of the cases it rapidly rises to  $104^{\circ}$  or  $105^{\circ}$  F. ( $40.5^{\circ}$  C.), but soon falls to  $102^{\circ}$  or  $103^{\circ}$  F. ( $38.8^{\circ}$  or  $39.4^{\circ}$  C.), at which level it is maintained with irregular undulations until defervescence, which takes place by lysis. In fatal cases death is preceded by a sudden great elevation of temperature to  $108^{\circ}$  and even  $110^{\circ}$  F. ( $43.3^{\circ}$  C.). There may be a rapid fall of temperature, followed by collapse. In the very young the thermometric range is lower than in adults.

The *pulse* is but slightly accelerated, if at all, in the early stages of the disease. Later, in twenty-four to thirty-six hours, it may in severe cases leap to 120 or even 140, its chief characteristic being the variability in its rate. In the early stage it is of good volume and tension; later, it may be soft and compressible, and when a fatal termination is impending it becomes small and feeble.

The *respirations*, as a rule, increase in frequency and are sometimes quite irregular; but marked dyspnea, with slowing of the respirations, may be observed during the advanced stage, being due to pressure exerted by the exudation upon the respiratory center. Cheyne-Stokes breathing and sighing respirations may be present.

**Nervous Symptoms.**—The *headache* is racking and often persistent, though it is subject to remissions; and is intensified by light and sounds, being so violent as to cause the patient to groan even while profoundly comatose. There is vertigo in nearly all instances. The pain referred to the spine may be general or limited to either the lumbar or cervical region (rarely the dorsal), and the general myalgic pains are often intense, especially in the extremities and the abdominal region. With the cephalalgia and abdominal pain may be associated vomiting. *Hyperesthesia* is a prominent symptom, the gentlest touch being extremely painful; *anesthesia* may also be noted, though less frequently, and usually follows the hyperesthesia. Any voluntary muscular movements, however, excite pain. In some cases *delirium* appears early, and in others rather late, while in the worst types death often occurs before delirium develops. On the other hand, in a small percentage of cases this symptom is absent throughout the entire course, and always its character and intensity exhibit a remarkable variety. It may be mild or it may take the form merely of incoherent answers to questions. Active delirium, however, is common and is accompanied by hallucinations, during which the patient shouts loudly, and, unless restrained, gets out of bed. This form of delirium occurs in paroxysms that are most apt to appear at night, and in the female it is sometimes hilarious or hysteric. An erotic tendency, with priapism or seminal emissions, has rarely been observed in males. The "maudlin" delirium of the drunkard is sometimes seen, but sooner or later somnolence appears and may deepen quickly into coma, the latter symptom perhaps being temporary, though more often it continues until recovery or death. As before stated, vomiting is common, though it may appear late in the disease; it is doubtless of cerebral origin.

*Symptoms of motor irritation* are among the prominent phenomena of



the disease, twitching of single muscles or groups of muscles often being seen, and occasionally muscular tremors. Muscular contraction is an almost constant feature. After a few days a tonic spasm of the muscles of the extremities sets in, as the result of which the arms are bent upon the chest, the forearm upon the arm, and the thumb upon the palm; the thigh is also flexed on the abdomen and the leg on the thigh. The opisthotonos previously alluded to may be followed by trismus, which can be considered a mortal symptom. Convulsions do not occur in adults, but are common in children; occasionally, however, there are paralysis, especially of the muscles of the face, and paretic hemiplegia.

**Organs of Special Sense.**—Photophobia is a prominent symptom, and the condition of the pupils is very variable. They may be dilated or contracted (more frequently the former) or remain normal; and in the majority of cases they are unequal in size and react poorly to light. These pupillary changes may come on early or late. Strabismus is frequent, being usually temporary, though it may recur several times during the attack. Rarely it is permanent. Conjunctivitis of moderate intensity and keratitis may occur, the former being more frequent than the latter, however; and ptosis is almost always present. Intense purulent irido-choroiditis sometimes occurs; either temporary or permanent blindness is met with, and, much more rarely, nystagmus is noted. Among optical sequelæ are cataract and atrophy of the eyeball.

*Deafness* is by no means an infrequent symptom, there being an early intolerance of sound and a marked tinnitus aurium. Later, suppurative inflammation of the middle ear, followed by rupture of the tympanum and otorrhea, may occur. The internal ear may be similarly involved, and in such cases the gait may become uncertain from implication of the semicircular canals. The deafness may after recovery be found to be permanent, though, as a rule, it is incomplete.

**Cutaneous symptoms** appear, some of which possess considerable diagnostic worth. Pallor and lividity of the skin and visible mucous membranes often characterize the period of invasion, and shortly after the onset herpes facialis appears in more than half the cases. This symptom is significant for diagnosis. The separate lesions are extensive, and often coalescence of two or more is witnessed. Herpes facialis belongs in a peculiar sense to cerebro-spinal meningitis—herpes labialis to malaria, and less frequently to pneumonia and meningitis. A petechial eruption is common, and has been most frequently met with in the early epidemics, and more frequently in America than in Europe. To this symptom the disease owes the name, long since given to it, of “spotted fever.” It may, however, be absent, and when present it is sometimes limited to a small superficial area, though more frequently it is diffuse. At first the eruption may be bright-red (erythematous), later becoming darker, or it may be distinctly petechial from the start; purpuric spots of considerable size and sometimes large ecchymoses may appear, but these are most common in the more malignant types. Other forms of eruption are also seen (sudamina, urticaria, ecchyma, erythema, erysipelas, etc.), but are devoid of diagnostic value. Gangrene of the skin is occasionally noticed, and in some cases bed-sores are liable to arise; but there is no fixed time for the skin-lesions of cerebro-spinal fever to appear, and their duration is exceedingly variable.



Of **gastro-intestinal symptoms** vomiting is the most common. It usually lasts only for a brief period at the onset, though it may recur later at longer or shorter intervals, and is of nervous origin. The appetite may be good, but in many cases it is soon lost, the tongue, in a large proportion of the instances, being only slightly coated. In cases assuming the adynamic or typhoid type the tongue is apt to become dry and of a brown color, with the formation of sordes. Under these circumstances the abdomen is tympanitic and the bowels relaxed, and diarrhea may be urgent, resisting all efforts aimed at its relief. Retraction of the belly is common, and *constipation* instead of diarrhea is the general rule; the spleen may often be felt a little distance below the costal margin.

**Renal symptoms** are not prominent, though the amount of urine passed is often above the normal despite the febrile movement. It may be below, though rarely, while in still other cases it is found to be about normal; and retention on the one hand and incontinence on the other have been observed. Albuminuria is sometimes met with, and sugar has been detected in the urine in rare instances.

**Complications.**—Many of these have already been mentioned in the portrayal of the symptoms, particularly those taking the form of destructive inflammations of the eye and ear and the paralyses of the cranial nerves. The purulent inflammations of the serous sacs which were referred to in discussing the pathology (pleurisy and pericarditis) are among the frequently associated conditions, and secondary bronchitis is also common.

*Pneumonia* (lobar and lobular) is a frequent and much-dreaded complication. Atelectasis may occur.

*Hemorrhagic nephritis*, usually of mild type, may appear as a complication.

**Special and Atypical Forms.**—(1) **Mild or Rudimentary.**—In this type the characteristic signs are either undeveloped or wanting, and the diagnosis is possible only during the prevalence of epidemics, which furnish typical cases. The symptoms vary and are indefinite, but perhaps the most constant and significant are severe headache, languor, vertigo, nausea, and occasionally vomiting. Fever and contraction of cervical muscles are absent, as a rule. The duration of rudimentary cerebrospinal fever is brief, the more noticeable symptoms rarely exceeding three or four days.

(2) **The Abortive Form.**—Here the initial symptoms are severe, but after two or three days they rapidly subside, leaving the patient convalescent. The disease is cut short by the acquisition of immunity, and not as the result of medical interference.

(3) **Intermittent Form.**—In this variety the symptoms, however intense, remit or almost wholly intermit every day or second day; these remissions are followed by a decided exacerbation or recurrence of the distressing features of the disease. Intermissions may occur at the beginning of a case, though more often they occur at an advanced stage and tend to prolong its course. There is not observed the strict periodicity that is seen in malaria, and neither is the temperature-curve typical of the latter disease nor are the malarial organisms found in the blood.

(4) **Typhoid Form.**—In a certain though small proportion of the cases

the special features are characteristic of the "typhoid state," but their course is more protracted than is usual.

(5) **Fulminant or Apoplectic Form.**—The symptoms characterizing this most malignant type of the affection are rather inconstant. There may be severe chill, loss of consciousness, followed by deep coma and death, the whole course occupying the space of a few hours only. I saw two such cases in the same family: the first, a girl of five years, was stricken at 2 P. M. and died at 9 P. M.; the other, a boy of seven years, was taken ill on the following day about the same hour, and died at 10 P. M. Other instances pursue a somewhat slower course, though manifesting the most striking malignancy. These begin with intense chills, violent headache, vomiting, early stupor, great prostration, contraction of muscles of the neck, moderate fever, and a feeble, progressively slowing pulse until it sometimes reaches 50 or even 40 beats per minute. The eruption, when it appears, takes the form of purpura. This form is most apt to be met with early in an epidemic, and with few exceptions proves fatal.

**Diagnosis.**—The most important symptoms for diagnosis are the abrupt onset; intense pains (cervico-occipital and lumbar); prostration; vomiting; vertigo; somnolence, alternating with local or general tonic or clonic convulsions; delirium (often sportive in type); tonic contraction of the muscles of the neck, extending to the back; marked hyperesthesia; a slow, followed by a more rapid though variable, pulse; irregular temperature-curve; and certain eruptions, especially petechial and herpetic.

**Differential Diagnosis.**—The disease, especially the sporadic form, is apt to be confounded with certain other affections.

(1) **Tubercular Meningitis.**—In this affection there is usually a tuberculous history—either personal or family—with prodromes extending over many days (occasional vomiting, unnatural peevishness, constipation, etc.). The invasion-period lacks the sudden onset of cerebro-spinal meningitis; the degree of retraction of the abdomen is greater than in the latter disease, while the arching of the neck is less; the general myalgic pains and the hyperesthesia are also less marked than in cerebro-spinal fever; the herpetic and petechial eruptions are rare in tuberculosis and common in cerebro-spinal meningitis; while Cheyne-Stokes breathing and the well-marked changes of pulse belong peculiarly to the tubercular type. By the aid of the ophthalmoscope choroidal tubercles may sometimes be detected, and are signs of an invariably fatal complaint.

(2) **Pneumonia.**—This affection may be complicated with a meningitis that affects chiefly the cerebral cortex. Hence, while there will be motor spasm (more or less localized) and tremors, there will also be less retraction of the head and less myalgic pain than in cerebro-spinal meningitis. Again, pneumonia precedes the development of the meningeal symptoms, and when not seen early we cannot be certain in sporadic cases which was the prior affection.

(3) **Typhoid Fever.**—The cerebral type of this affection may simulate closely the disease under consideration. In both may be observed fever, delirium, somnolence, retraction of the neck, spasm, tremor, and profound prostration. The mode of onset, however, is different, being slower in typhoid and unaccompanied by vomiting, muscular spasm, or hyperesthesia. In typhoid there is also the characteristic mental dulness; the



fever is higher, with a typical temperature-curve; the roseate eruption is characteristic, appearing in crops at a definite time and runs a definite course; and there is greater enlargement of the spleen.

**Sequelæ.**—The leading sequelæ are permanent blindness (due to optic neuritis with atrophy) and deafness, which sometimes terminates in deaf-mutism; and in many cases headache outlasts the disease for months or even years. Chronic hydrocephalus and mental enfeeblement are not rare sequels (Ziemssen). Various local paralyses are observed, affecting either single extremities or single groups of muscles or the muscles supplied by the different cranial nerves, and recovery is to be expected in these cases after a few months. They are most probably due to certain peripheral lesions (neuritis and perineuritis).

**Immunity.**—Permanent immunity is rarely conferred by the occurrence of cerebro-spinal meningitis, *relapses* being common, and *second* attacks having been occasionally observed.

**Duration and Prognosis.**—In very mild forms the duration is from one to four or five days. The most malignant type runs an even shorter course, when, as is the rule, it terminates fatally. If recovery ensues, it is after a long, serious, and protean illness. The *abortive form* is necessarily of brief duration. In the ordinary type convalescence usually sets in at the end of one or two weeks, though not a few cases are met with in which the latter period is much delayed, and a slow convalescence, hindered by numerous complications and sequelæ, is the rule.

The *prognosis* is influenced especially by the degree of severity of the type. Apart from the *fulminant form*, which nearly always proves fatal, the severity of the infection may be appreciated by noting the degree of fever and the intensity of the nervous symptoms, especially the vomiting, coma, headache, opisthotonos, character of the respirations, etc. Complications may likewise affect the prognosis, pneumonia, and suppurative inflammations of the pleura or pericardium, rendering it particularly grave. Circumstances connected with the individual are also potent, and particularly the *age*. In children under two years the disease is very fatal, this period giving the highest mortality-rate; between two and five and after thirty years it is a more serious disease than during young adult life. The death-rate of cerebro-spinal fever varies greatly in different epidemics, ranging from 25 per cent. in the mildest to 80 per cent. in the severest.

**Treatment.**—(1) **General Management.**—The patient should be isolated, and the sick-room must be quiet and somewhat dark. All excitement is to be avoided; the patient must not be allowed to leave his bed until convalescence is firmly established; and the rules for preventing the spread of infectious diseases are to be strictly enforced.

The *diet* should be composed of nutritious liquids, such as milk and animal broths, etc., and as soon as convalescence begins the dietary should be increased by the addition of semisolid substances (rice, eggs, milk-toast, etc.), and, finally, the more easily digestible solids. The general course, and particularly the period of convalescence, may be much abridged by the systematic administration of appropriate articles. Water must be offered to the patient frequently.

**Medicinal Treatment.**—Many and widely various modes of treatment have been recommended by as many different authors, but in my opinion it is best to treat individual cases according to the special indications pre-



sented. I regard it as extremely improbable that any case of this affection has been benefited by venesection. Cold or gradually cooled baths, when the handling of the patient does not excite too much pain, are of great value, and warm baths will prove highly beneficial by lessening the tendency to tonic spasm of the muscles.

Among medicinal agents narcotics are the most useful. Morphin, particularly when administered hypodermically, affords prompt relief from intense headache, myalgic pains, muscular contraction, and other nervous symptoms, and at the same time spares the heart. If the respirations be irregular, atropin may be combined with the opiate, and if the heart threatens to fail, strychnin may be administered. In young children we must rely upon the bromids rather than the opiates, and the former are quite effective in young subjects. In older children we may employ opium if we do so cautiously, and I have found the deodorized tincture of opium and paregoric to be the best preparations under these circumstances.

For the tonic contraction of the muscles, especially when associated with violent cerebral symptoms, cannabis indica should be tried. Convulsions call for warm baths or ether-inhalations. Mercury has been, and still is, firmly advocated by certain authors, and, cerebro-spinal meningitis being an infectious disease, this drug may be given for its antiseptic virtue (mercuric chlorid gr.  $\frac{1}{24}$  (0.002) every four hours to an adult; calomel, gr.  $\frac{1}{12}$ — $\frac{1}{16}$  (0.005–0.004) every four hours to children). Belladonna and ergot have been employed to diminish the congestion of the cerebro-spinal capillaries. They should be administered in the early stages, and, thus employed, I have found them in my experience not wholly without value. Antipyrin, acetanilid, and phenacetin are not to be thought of in the treatment of this disease, owing to their depressing effect upon an already overburdened heart.

**Stimulants** are required if signs of heart-exhaustion appear. They may be freely exhibited in accordance with the rules that obtain in other acute infectious diseases.

During the advanced stage or after effusion of the exudate has taken place the narcotics are to be replaced by agents that promote absorption, and particularly the potassium iodid in full doses.

The **local means** are also important. When tub-baths are not available, cold should be used locally, since it is both of value and very grateful to the patient. An ice-bag is to be put on the head, and, if possible, long ice-bags placed along the spine. In rare cases of asthenic type we may employ small blisters at the nape of the neck or over the mastoids: these should be applied early, though they are also useful during the stage of effusion. In the usual form of the disease it is better to apply the thermo-cautery lightly over the mastoid region. If the patient be not too much enfeebled, we may abstract a small amount of blood by means of leeches or by a few wet cups placed behind the ears.

**Convalescence** is prolonged, and requires to be diligently and judiciously treated. We must rely upon the generally accepted tonics—iron, cod-liver oil, arsenic, and strychnin; the potassium iodid and the mercury also being continued for their influence in promoting the absorption of the exudate. Special attention is, however, to be paid to the hygienic management of this period. An abundance of fresh air, sun-

shine, and easily assimilable food must be furnished at all hazards, and electricity and massage, judiciously employed, will hasten recovery.

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## LOBAR PNEUMONIA.

(*Croupous or Fibrinous Pneumonia; Pneumonitis; Lung Fever.*)

**Definition.**—An acute infectious disease caused by the *Micrococcus lanceolatus*, which produces a specific inflammation of the parenchyma of the lung and marked constitutional disturbances—chill, extreme prostration, and fever which terminates by crisis. Secondary septic complications are frequent. There are different forms of lobar pneumonia, classified according to their clinical or pathologic peculiarities, as primary lobar pneumonia, secondary lobar pneumonia, and lobar pneumonia with the formation of new connective tissue, etc. I shall describe the first two forms under the present heading, and the third separately (p. 510).

**Pathology.**—Usually the lesions are confined to the whole of one lobe; less frequently to the whole of one lung, and rarely to parts of both lungs. From Jürgensen's analysis of 6666 cases the following statement, showing the different situations of the lesions and their relative frequency, was taken: Right lung, about 54 per cent.; left lung, about 38 per cent.; and both lungs, about 8 per cent. In the right lung the lower lobe was involved in 22 per cent., the upper in 12 per cent., the middle in nearly 2 per cent., and the whole lung in about 9 per cent. In the left lung the lower lobe was involved in about 23 per cent., the upper in about 7 per cent., and the whole lung in about 8 per cent. Both lungs were implicated in 8 per cent.

The lesions of pneumonia are those of three stages: (a) Stage of congestion or engorgement; (b) Red hepatization (consolidation); and (c) Gray hepatization.

(a) **Stage of Engorgement.**—The part or parts implicated are of a dark-red color, and firmer to the feel, but less resilient and crepitant, than normal. The cut section drips a blood-stained serum, and dark blood exudes from the distended capillaries. The air-cells do not collapse, though they are not solid, since excised pieces float; but the weight of the lung-tissue is much increased and the air-sacs are distended with the corpuscular exudate. Collapsed portions may be observed which may readily be insufflated from the bronchus, and areas of extravasation may occasionally be noted near the pulmonary pleura.

On microscopic examination the alveolar epithelium is seen to be swollen, the capillaries greatly distended, and the air-cells filled with alveolar epithelial cells, red corpuscles, and a few leukocytes. Similar elements occupy the small bronchi, while the mucosa of the larger bronchi is often hyperemic.

(b) **Red Hepatization.**—The affected tissue is solid, airless, and firm, resembling, as the term indicates, liver-tissue. It is reddish brown (mahogany) in color, presenting a dry, mottled appearance, and when, as is usual, an entire lobe is involved, it is more voluminous than normal and



its surface is often furrowed by the impress of the ribs. Being airless, the affected portion does not crepitate, and its weight and specific gravity are increased. It cannot be inflated; is extremely friable, and its lacerated surface presents a finely granular aspect, this latter appearance being due to the minute plugs of inflammatory matter (fibrin) which fill the air-spaces. The air-passages and small bronchi are distended with similar material, and granular masses can be removed from the air-cells of a cut or lacerated surface by carefully scraping the latter. If death takes place during this stage, the ante-mortem, dry, inflammatory exudate soon softens, and may flow from the cut section as a grumous, viscid fluid; the consolidated tissue sinks rapidly in water. The pulmonary pleura is covered with a fine sheet of fibrin, and in cases preceded or complicated by marked pleurisy the fibrinous, inflammatory exudate forms a thick coating upon the pleural membrane, and the sac contains more or less liquid effusion (pleuro-pneumonia).

Microscopic examination shows the air-spaces filled with clotted fibrin, in whose meshes are held red blood-corpuscles, pus-cells, and changed alveolar epithelium. The interlobular connective tissue may be infiltrated with leukocytes and fibrillated fibrin, but the blood-vessels in the walls of the alveoli remain pervious. The pneumococci (*micrococci lanceolati*), less frequently also streptococci and staphylococci, are revealed by the microscope.

(c) **Gray Hepatization.**—In this stage the fibrinous exudation becomes decolorized, the surface at first resembling granite in color, and later appearing uniformly gray. Associated with this change, and following it, there is fatty and granular degeneration of the inflammatory exudate, in consequence of which the latter becomes moist and soft. The exudate loses its granular character, while at the same time the friability of the lung-tissue is further increased, and from the surface of the cut section there flows usually a grayish-white or yellowish-white purulent liquid. Not less than one-half of the fatal cases die in the early part of this stage. The pleura that invests the involved tissue is usually covered with a fine fibrinous exudation.

Microscopic examination shows the air-cells stuffed with leukocytes, while the other histologic elements (fibrin, red blood-cells, etc.) have disappeared; and the full development of gray hepatization marks the beginning of resolution, though the latter process may in reality begin with the commencement of the former. The exudate is now softened into a liquid material, with disintegration of cellular elements, and is absorbed by the lymphatics. *Resolution* usually corresponds in time with the occurrence of the crisis, though it may begin later. Again, the process may be much prolonged.

Among unfavorable terminations may be noted—

(1) **Purulent Infiltration.**—Here the lung-tissue becomes very soft, friable, and is bathed in purulent material; and microscopic observation shows the pus-cells densely infiltrating the interalveolar tissue and filling the air-spaces as well. This impairs the nutrition of the lung-tissue, and may thus cause rupture of the septa, producing

(2) **Abscess.**—This is to be attributed to subsequent infection by streptococci, and hence is a complicating lesion. The abscesses vary in size within the widest limits, most frequently being situated near the base



of the lung, and may occupy the periphery and rupture into the pleural sac, causing pyo-pneumothorax. In most instances the abscess-cavity has a fistulous connection with a bronchus, but occasionally the abscesses become encapsulated in fibrous tissue, their contents undergoing first caseous, and then calcareous, degeneration. Rarely they open into the pericardium, and still more seldom externally. They may be small and multiple, in which case they sometimes coalesce, forming large abscesses.

(3) **Gangrene** may rarely follow, but is due to a specific cause, and hence does not belong especially to the pneumonic process.

(4) **Induration**.—A. Fränkel states that in a few instances (about 1 per cent.) pneumonia ends in induration, and is found upon section to be smooth and its tissue resistant. The surface of the cut section sometimes shows a peculiar transparency, with characteristic yellow specks, due to the collection of cells which have become fatty.

Microscopically, the alveoli are seen to be blocked up by connective tissue resembling polypi and containing vessels. By its structure it recalls the process of organization in a thrombus, and is probably due to secondary infection with a specific bacillus. It may also be observed after broncho-pneumonia.

**Changes in Other Viscera**.—The *heart* often appears pale and is flabby, but upon microscopic examination the muscular cell-fibers of the organ are not found to be degenerated, except in rare and usually protracted cases. The cardiac chambers, particularly the right, are distended with firm, tough clots, which are usually removable *en masse* from the great vessels in the form of arboreal casts. To account for the great tendency to coagulation of the blood in pneumonia is the fact that its fibrinous elements are vastly increased.

*Pericarditis* occurs in about 5 per cent. of the cases, and is relatively more frequent in left-sided or double pneumonia. *Endocarditis* is more common, especially the ulcerative form, which was present in 11 out of 100 autopsies (Osler). With malignant endocarditis the lesions of meningitis are often combined, but as a separate complication meningitis is rarely encountered.

The *spleen* is congested, moderately enlarged, and softened, and the *liver* is likewise hyperemic and somewhat swollen. In the *kidneys* are found the lesions of parenchymatous inflammation, and with remarkable frequency also those of chronic interstitial inflammation. A catarrhal state of the gastro-intestinal mucosa (often with jaundice) is common; and a frequent complicating change is croupous inflammation of the colon. A true diphtheritic colitis, however, occurs but seldom.

**Etiology**.—**Bacteriology**.—The generally accepted specific cause of pneumonia is the *Micrococcus lanceolatus* of Fränkel. It is a lance-shaped (slightly elliptic) coccus, united in pairs (a fact to which it owes its name of diplococcus), and is present occasionally in the nose, Eustachian tubes, and larynx of healthy individuals. Netter found it in 20 per cent. of the specimens of buccal secretion taken from well persons, and to the presence of this germ is to be ascribed the form of septicemia induced in animals by inoculation with saliva. It is present in about 90 per cent. of all instances of pneumonia, and in persons who have had the disease it is detectable for many months or even years. It is generally present in pure culture, but may be associated with pyogenic organ-

isms. It is probable that Friedländer's bacillus and other micro-organisms may also have the power to cause the disease; and Wassermann<sup>1</sup> suggests that specific forms of pneumonia may coexist in the same individual, as, for example, lobar pneumonia and influenzal pneumonia, the latter being due to the bacillus of Pfeiffer. The *Micrococcus lanceolatus* (Fig. 15) can be readily demonstrated in the sputum by treating a cover-

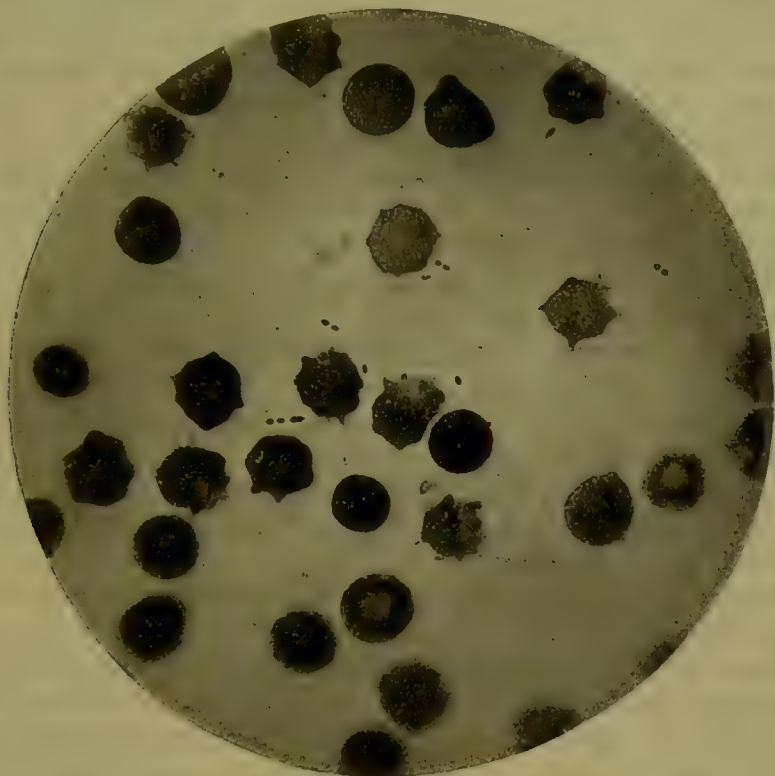


FIG. 15.—*Diplococcus pneumoniae*, from the heart's blood of a rabbit;  $\times 1000$  (Fränkel and Pfeiffer).

slip preparation "with glacial acetic acid, and then, after washing off the acid, dropping on anilin oil and gentian-violet, which is to be poured off and renewed two or three times."

The mode of infection is not positively known, but it is highly probable that the pneumococcus is inhaled. The first and chief effects of the germ are local—in the lung, though it may reach more distant portions of the body, such as the pleura, pericardium, endocardium, meninges, etc., and the latter structures may rarely be invaded in the absence of involvement of the lung. To the widespread distribution of the pneumococcus is due, in part, the septicemic process sometimes observed. Usually, then, the disease is a local one at the start, but soon the toxins of the *Micrococcus lanceolatus* become diffused throughout the system, producing a general disturbance. *Secondary infection* with other specific organisms (streptococci, staphylococci, etc.) commonly occurs in the various organs of the body.

**Predisposing Causes.**—(1) **Endemic Influence.**—Among the populace of a community sporadic cases constantly occur, although persons leading an out-of-door life in rural districts are less susceptible to the disease than are residents in cities. That epidemics of pneumonia, often of

<sup>1</sup> *Deutsche medicinische Wochenschrift*, Leipzig, Nov. 23, 1893.



serious type, may occur in solitary buildings (barracks, tenement-houses, institutions, etc.) cannot be successfully denied, and here the disease appears to make for itself, rarely, a permanent home. We may, with justice, attribute these outbreaks to defects in local sanitary conditions, which favor the propagation of the specific agent and tend to lower the bodily resistance to bacillary invasion.

(2) **Epidemic Influence.**—From time to time pneumonia prevails extensively, and appears to spread throughout a considerable percentage of the entire population of urban and rural districts. It may also originate in the endemic form in tenement-houses and institutions, and increase in its scope until it assumes an epidemic character. The epidemic form of pneumonia is at times confined to private homes (house epidemics), and in the winter of 1894 I saw, with Dr. W. K. Mattern of Philadelphia, 3 cases develop in rapid succession in one family. A Sister of Charity, after nursing two of the patients faithfully for a period of ten days, was also attacked and died of the disease. There are many similar instances on record in which several members of the same family have contracted the disease at about the same time, and it is possible that the house-epidemic form may spread by contagion. An instructive epidemic is reported by W. B. Rodman, who states that 118 cases of pneumonia, with 25 deaths, occurred in a prison population of 735; and numerous epidemics of similar character have been observed in other localities, both at home and abroad.

(3) **Geographic Distribution.**—Pneumonia may be said to be an almost universally distributed affection. It prevails, however, more extensively in certain countries than in others, and occurs more frequently in certain sections of the same country than in others. Thus, Delafield<sup>1</sup> points out the fact (based on the eighth and ninth census reports) that in the United States the disease is of more frequent occurrence in the South than in the North. Climate, *per se*, does not, however, exercise a notable influence.

(4) **Season.**—Of 5905 cases collected by Seitz in Munich, 36.8 per cent. occurred in the spring, 32 per cent. in winter, 15.7 per cent. in autumn, and 15.3 per cent. in the summer. The period of maximum frequency of the affection in temperate climates is usually from the beginning of February to May, inclusive, and the next most frequent period is from December until February. In London most cases appear between the end of March and the end of June (Herringhan). The period of greatest frequency will be found to correspond in time with the period of the greatest vicissitudes of temperature and humidity, though it cannot be affirmed positively that there is an essential connection between the latter condition and pneumonia. Richter<sup>2</sup> claims that when the atmospheric pressure is high the cases are more numerous; while, on the other hand, when it is low the cases are much fewer in number.

(5) **"Catching cold"** is often followed by pneumonia, but frequently there is no such history. In this condition the mucosa of the respiratory passages is so altered as to become more susceptible to infection with the pneumococcus, and hence the so-called "cold" is a predisposing cause.

<sup>1</sup> "Diseases of the Lungs," *American Text-Book of the Theory and Practice of Medicine*, Pepper, vol. ii. p. 540.

<sup>2</sup> *The Journal of the American Med. Assoc.*, Aug. 4, 1894, p. 188.



Such facts as these also explain why pneumonia occurs with undue frequency in persons following certain occupations exposing them to those external influences that are apt to excite "cold."

(6) **Traumatism.**—Following injuries, especially of the chest, pneumonia occurs quite frequently. Contusions of the thorax by lowering the vital power and resistance of the tissues probably produce the same local effects as taking "cold."

(7) **Age.**—Lobar pneumonia is common at all periods of life, and during the first two years of life lobar pneumonia is quite frequent. Between two and twenty years of age there is less liability, and between twenty and forty it is again increased; while from forty to sixty years susceptibility again diminishes. After the latter period it augments rapidly.

(8) **Sex.**—Males are, on the whole, more frequently attacked than females, the discrepancy in the relative number of cases being greatest from the twentieth to the fiftieth years of age, and being due to the different degrees of liability to exposure in the two sexes.

(9) **Unhygienic Surroundings.**—The disease is more frequent among the lower than the higher classes—a fact due to the improved hygienic surroundings of the latter, since, doubtless, anything that will lower the vital energy will serve as a predisposing factor.

(10) **Circumstances connected with Individuals.**—The alcoholic is especially prone to this disease, any or all habits that tend to depress the nervous system acting as predisposing causes. Certain chronic diseases may exert an influence (chronic Bright's disease, organic heart-affections, carcinoma, diabetes, etc.); but, contrary to what is observed in other acute infectious diseases (typhoid fever in particular), susceptibility is not so great among immigrants and new-comers as among the natives and the older residents.

(11) **Prior Attacks.**—One attack undoubtedly leaves the system more susceptible to the disease, so that repeated attacks may occur in the same individual. And yet while it is true that persons have had numerous attacks—ten or more—this predisposing influence has probably been overestimated by most writers.

**Immunity.**—The results of the investigations of Behring and Kitasato with the blood-serum of animals which had been immunized against tetanus and diphtheria led Drs. G. and F. Klemperer to experiment upon the lower animals with Fränkel's diplococcus. They found that the rabbit could be rendered immune by intravenous or subcutaneous injections of large amounts of the fluid bouillon-cultures or of the glycerin-extract. From 10 to 20 c.c. of serum taken from a non-receptive animal were injected into the veins of an animal that was suffering from typical pneumonia (artificially produced), whereupon the symptoms subsided rapidly and the animal entered upon a speedy recovery. The same serum, used in a similar manner upon healthy receptive animals, rendered them non-receptive. The important truth that the serum of the blood of patients during convalescence from pneumonia contains an antitoxin which, when injected into the venous system of infected animals, is found potent to cut short the disease, has also been demonstrated by these observers. They have employed the blood-serum of pneumonic patients *after* the crisis, injecting it into other patients *before* the crisis with a view to

inducing the latter, and success has attended their efforts in 6 cases. The question of serum-therapy for this important affection in man is not finally cleared up, and is still beset with difficulties; but that the pneumococcus engenders a virus—pneumotoxin—which produces elevation of temperature, etc. has been clearly demonstrated by the Klemperer brothers. Again, that this substance, acting upon the albuminous elements of the body, generates an antipneumotoxin which circulates in the blood and neutralizes the pneumotoxins as they are formed, inducing the crisis, has also been clearly proved. Antipneumotoxin, however, has not as yet been isolated.

**Clinical History.**—*Prodromes* are rare, and when present consist merely of a slight general indisposition, lasting a day or more. Rarely, there is cough, thoracic oppression, and slight chest-pains (simple bronchitis), that may or may not be connected with the pneumonic process. When this is the case, however, the invasion may be marked by sudden, great thoracic oppression or by a gradual development of the local and general symptoms.

Usually the onset is very abrupt, being marked by a severe rigor, which has a duration of from half an hour to an hour, during which period the patient feels most uncomfortable, and is, indeed, very ill. The initial chill may occur at any hour of the day or night, the fever rising immediately and rapidly, and the temperature often mounting to 104° F. (40° C.) or even higher in the course of a few hours. The skin becomes harsh and dry, the face flushed, and the cheek on the side affected often shows a circumscribed deep-red spot. Prostration is pronounced, and headache and other nervous disturbances (restless delirium, etc.) accompany and follow the ushering-in symptoms.

The *thoracic symptoms* follow closely upon the termination of the chill. Inspiration, particularly if deep, causes a stabbing pain in the affected side; the respirations are hurried, somewhat jerking and shallow (panting), while the pain persists, and later dyspnea may become marked, with accelerated breathing. Cough sets in early, and is dry and painful during the first day or even longer, and may be attended with expectoration, which generally presents a characteristic *rusty* or *blood-stained appearance*. The physical signs rarely appear before the end of the first day, and sometimes as late as the third (central pneumonia); in the latter form the local symptoms, as cough, dyspnea, and sometimes pain, are either wanting or feebly marked during the first three or four days, and the clinical picture is composed of the general features only.

Anorexia is usually complete; thirst is excessive, and there may be vomiting at the onset, the bowels being generally constipated, though diarrhea may not infrequently be present. The patient in most instances lies upon the affected side until the pain has in great part subsided, and then he is apt to assume the dorsal position, exposing to full view an anxious countenance, with a characteristic flush upon the cheek, while the *alæ nasi* are seen to dilate forcibly during inspiration. Very frequently herpes on the lips or nose appears about this time, and forms a valuable diagnostic symptom. The nocturnal remissions are slight, the temperature being of the continued type, and the fever continues high—104° to 105° F. (40.5° C.)—for from five to ten days, and generally terminates by crisis. The pulse is somewhat quickened, but



the pulse-respiration ratio is not maintained. The other general features last until the crisis occurs, or even increase in severity, but do not outlast this period; many of the local symptoms, however, and particularly pain, are greatly improved before the crisis is reached.

As will be seen hereafter, the general course of pneumonia is modified by a variety of interfering conditions that have relation to complications, individual circumstances, severity of the type, etc. In the instances in which the crisis is reached convalescence is rapidly established. The crisis may be accompanied by special symptoms, as copious sweating or diarrhea.

**Leading Symptoms in Detail.—Local or Respiratory Symptoms.**—Increased frequency of the respirations is a characteristic symptom, the rate varying from 40 to 60 per minute in adults, and in children from 60 to 90 or more. It is panting in character, particularly when pneumonia occurs in old subjects, and both inspiration and expiration are brief, though sometimes separated by a rather long pause. Expiration is usually accompanied by an audible “grunt,” indicating great oppression, and while actual dyspnea is a frequent symptom, it may be absent or as the case progresses may become either increased or greatly diminished according to the severity of the type.

The *chief causes* of the rapid and labored breathing are the involvement of a large portion of the lung, associated severe general bronchitis, pericarditis or extensive pleurisy, cardiac failure, collateral congestion with edema, fever, and the intense pain in the side.

The pulse-respiration ratio is disturbed, the relation now being 1 to 2, or even 1 to 1.5, instead of 1 to 4, as in health (see Fig. 16).

*Pain* in the affected side is in most cases developed within a few hours after the initial chill, and after lasting two or three days gradually disappears. It is stabbing in character, and usually referred to the region immediately below the nipple or to the axilla, and rarely to other points (abdomen, flank, etc.). In most instances it is not severe until greatly intensified by the cough, which always aggravates this symptom, as does deep inspiration. The pain is due to implication of the pleura covering the inflamed lung, and may be entirely absent, though usually in the aged only.

The *cough*, like the chest-pain and respiration, is somewhat characteristic, being frequent, short, dry, and voluntarily repressed, because it is attended with increased suffering. Yet there are cases that run their entire course without cough, and this especially in the aged and in drunkards.

*The Sputum.*—At first mucoid and frothy, it soon becomes of a characteristic *rusty* color. It consists of a frothy, fluid mucus containing an abundance of small viscid masses of a yellowish- or reddish-brown color, from admixture of blood. The chief peculiarity of the sputum in fully developed cases is its *viscosity* and *tenacity*, often adhering to the receptacle even though the latter be inverted; owing to its adhesive quality it is ejected from the mouth with considerable difficulty by the patient. About the time of the crisis the sputum usually becomes more abundant, distinctly purulent, and its expulsion easy, but rarely it may be absent after the crisis. In severe types of the disease it may, at the outset, consist largely of pure blood, and in adynamic forms it is often



thinner and darker in color (*prune-juice*). There are cases in which there is an abundance of muco-purulent expectoration when extensive associated bronchitis occurs, and, on the other hand, instances are met with in which nothing is expectorated save a little light-colored mucus. In old persons or in those previously enfeebled there may be no expectoration whatsoever. The amount is therefore exceedingly variable, not only in different cases, but also in different stages of the affection.

Under the microscope the sputum is seen to contain red blood-corpuscles, alveolar epithelium, the *Micrococcus lanceolatus* (usually with other micro-organisms), pus-corpuscles, and small fibrinous casts.

**General Features.—The Fever.**—As I have already stated, the fever rises rapidly during the initial chill, so that in eight to twelve hours the temperature reaches  $104^{\circ}$  or  $105^{\circ}$  F. ( $40.5^{\circ}$  C.). It then remains high until the crisis, pursuing the continued type, with nocturnal remissions amounting to a degree or over, while the daily fluctuations correspond with the normal, except that they are now somewhat exaggerated. In children the rigor is almost always replaced by convulsions. The temperature has a lower average range in persons previously debilitated, in old people, and in drunkards, than in healthy adults and children. During the febrile period there may be observed a pronounced fall of temperature—pseudo-crisis—but the temperature again rises to its former height. This may occur quite early, though more often it precedes the true crisis by a day or two; and rarely it may take place repeatedly, and the temperature-curve bear a strong resemblance to the remittent or even the intermittent type, regardless of any malarial affection. The temperature may be unusually high,  $106^{\circ}$  F. ( $41.1^{\circ}$  C.) or even  $107^{\circ}$  F. ( $41.6^{\circ}$  C.), these striking elevations sometimes immediately preceding the crisis (*perturbatio critica*); but this does not belong particularly to pneumonia. It is especially characteristic of pneumonia, however, that the fever terminates by crisis; hence a mere glance at the temperature-chart may serve to complete the diagnosis in doubtful cases (see page 141). The crisis may occur anywhere from the end of the third to the fourteenth day, but in the majority of instances it is on the fifth or the seventh. The temperature usually falls during the night, and the drop is accompanied by copious perspiration, so that by the following morning the thermometer is found to register at the normal, or more often a subnormal, point ( $96-95^{\circ}$  F.— $35^{\circ}$  C.). This fall in temperature may also be interrupted by fresh though slight exacerbations.

The duration of the period of decline is usually from eight to twelve hours. It may be much shorter, but more often is much longer, just as when the decline takes place by lysis. The latter mode of termination is usually due to some complication, and when the high fever persists for an indefinite period (twelfth to fourteenth day or longer), it is usually due to delayed resolution.

**Circulatory Symptoms.**—Most important is it to study the condition of the heart and pulse in cases of pneumonia. The average pulse-rate in typical cases is about 100 to 108 per minute, and when it exceeds 120 there is just cause for alarm. The rate may be increased either suddenly or gradually, but in any event augmented frequency implies danger, since it is a certain indication of failure of heart-power. The latter may be due to the influence of the poison secreted by the diplo-

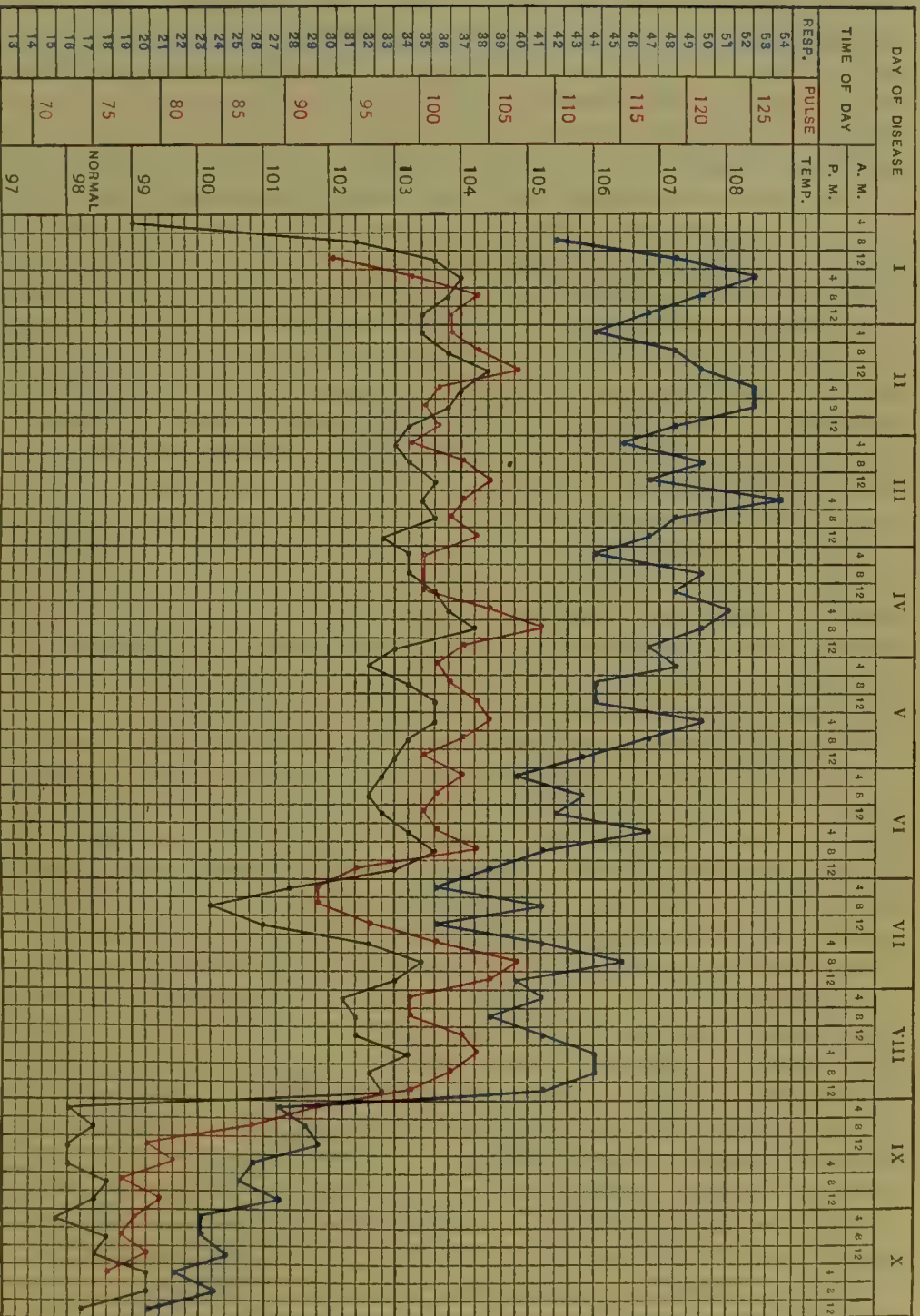


FIG. 16.—Chart of a case of lobar pneumonia with favorable course. A. T., aged thirty-two years; lower right lobe affected. Black, temperature; red, pulse; blue, respirations.



cocci, to previous organic disease of the heart, or to some complicating condition (pericarditis, collateral edema, etc.), and the period of greatest liability is in the advanced stage of the disease. At first the pulse is small, but a little later full and bounding. With complete and extensive consolidation the pulse is apt to become small, due to the fact that a lessened amount of blood reaches the left ventricle and the general systemic circulation. Dicrotism is sometimes noticeable, and an irregularity in the volume and rhythm of the pulse may be observed; it is an unpropitious sign. In the aged and the debilitated a small, feeble, and frequent pulse may be present throughout the attack.

The heart-sounds are clear, and owing to increased tension in the pulmonary vessels the pulmonary second sound is accentuated. This is the state of things throughout in favorable cases. With failure of the right ventricle (a not rare event) there arise the signs of dilatation of this chamber (extension of cardiac dulness to the right, epigastric impulse, a low systolic murmur, shortening of the diastole, or fetal heart-sounds, signs of venous stasis, and indistinctness of the pulmonary second sound). The strength of the right ventricle, upon which so much depends in pneumonia, is indicated by the character of the pulmonic second sound. A soft, low-pitched murmur is sometimes audible in the mitral and pulmonary zones.

The *blood-appearances* are somewhat characteristic. The researches of Lache<sup>1</sup> show that leukocytosis is of some value in determining between the crisis and pseudo-crisis, continuing in spite of the fall of temperature, etc. in pseudo-crisis, while it disappears with the true crisis.

Stienon<sup>2</sup> finds that in the febrile stage the polynuclear forms predominate, but as soon as these diminish the eosinophiles begin to increase.

The red corpuscles and hemoglobin remain little changed during the course of the disease, but show a marked decrease almost immediately after the actual crisis.<sup>3</sup> The prognostic significance of absence of leukocytosis would seem to be considerable, as this symptom serves to distinguish pneumonia from influenza, in uncomplicated cases of which it does not occur. The blood-plates are also increased in number (Hayem), and the micrococcus has been found in the blood, though rarely.

**Cerebral Symptoms.**—Headache sets in early and may be a prominent and persistent feature. In many cases, and particularly in children, the disease is ushered in by convulsions, this symptom occurring more often in the apical than in the basilar form of pneumonia. Delirium may come on during the acme of the disease and may assume a maniacal form, but more often in my experience consciousness has been retained during the whole attack in all but the severest cases. In the drunkard delirium tremens usually develops, and may anticipate the symptoms referable to the lungs; and I fully agree with Osler in stating that it should be an invariable rule, if fever be present, to examine the lungs in delirium tremens. These cases may often be appropriately termed “walking pneumonia,” since they go about until excitement gives way to a coma that deepens into death. In adynamic forms a low, muttering

<sup>1</sup> *Berliner klin. Woch.*, 1893, Nos. 36 and 37.

<sup>2</sup> *La Presse Méd.*, July 13, 1895.

<sup>3</sup> Sadler, *Fortschritte der Medicin*, 1892; Leichtenstein, *Ueber der Hämoglobin-gehalt des Blutes*, etc., Leipzig, 1892.



delirium is frequent, and is sometimes accompanied with more or less coma.

In the so-called *cerebral* pneumonia the nervous phenomena are quite pronounced, and simulate closely cases of cortical meningitis. It is often associated with excessively high fever, except in the aged, when the cerebral symptoms are also well marked, but the fever is moderate. Most authors contend that apical pneumonias are apt to assume the cerebral type, but, according to my own experience, this dictum is correct as relating to children only. Most authors also state that double pneumonias are characterized frequently by severe cerebral symptoms, yet I have seen several instances in the adult without unusual nervous phenomena.

**The Cutaneous Symptoms.**—As stated before, herpes is common and its diagnostic importance is considerable. Naso-labial herpes is but little less frequent in this disease than in malaria, being present in about one-third of the cases. It usually comes out from the second to the fifth day of the disease, and rarely may appear upon the cheek, lobe of the ear, the genitals, forearm, or upon the mucosa of the tongue. Sweats are not common during the height of the disease, but usually accompany defervescence, when they may be copious. The deep-red circumscribed spot upon one cheek (*mahogany flush*), usually on the side of the affected lung, has already been mentioned. Urticaria has been observed, though rarely.

**Digestive System.**—The mucous membrane of the mouth is dry, the tongue has a coating of a yellowish-white color, becoming dry and brown in cases representing a low form, and anorexia and thirst are present. Vomiting is not uncommon at the outset, and may be repeated, while constipation is the general rule and diarrhea the frequent exception. The above symptoms spring from the marked fever. Splenic enlargement of slight degree can usually be detected on palpation, but the liver is not perceptibly increased in size.

**Urinary Symptoms.**—The urine is febrile, diminished in amount, and high-colored, the urea and uric acid being greatly in excess. On the other hand, the chlorids are, according to the older authors, either diminished in amount or absent during the febrile stage, presumably for the reason that they pass into the inflamed lung-tissue. They are not, however, constantly absent, and sometimes they are not even lessened, in pneumonia; moreover, their disappearance is not peculiar to this disease. The above-mentioned facts justify two important inferences: (1) The absence of chlorids is a symptom of little diagnostic value; and (2) their reappearance in the urine toward the close of pneumonia is of small prognostic worth. Slight (febrile) albuminuria is common.

**Physical Signs.—Stage of Congestion.**—The density of the lung is increased, but the involved tissue is not consolidated and the pleura is not yet covered with fibrin.

**Inspection.**—The movements of the affected side (especially if the base be involved) are defective, the degree of expansion being much diminished. In double pneumonia the costal type of breathing, combined with a vigorous play of the abdominal muscles, is observed.

**Palpation.**—There is a slight increase in the tactile fremitus over the congested area.

*Percussion.*—The note may be normal, though more often it is briefer, higher-pitched, or even distinctly tympanitic.

*Auscultation.*—The breath-sounds are weak, and sometimes become broncho-vesicular upon deep inspiration, while over the unaffected lung-tissue they are exaggerated. If, as often happens, inflammatory products due to associated bronchitis occupy the small bronchi, subcrepitant râles may be audible. The crepitant râle, however, is rarely heard until the close of the first stage or until fibrin coats the pleural surfaces, and I cannot agree with the view of certain authorities who claim that this râle is produced in the air-cells and finer bronchi.

**Stage of Consolidation.**—*Inspection.*—There is little or no expansive motion of the chest over the affected area, while upon the unaffected side it is increased. The volume of the thorax on the diseased side is increased, as shown by mensuration, but the intercostal depressions are not effaced.

*Palpation* renders clearly perceptible the defect or absence of expansion. Vocal fremitus is usually much increased, though in exceptional instances it is diminished or absent—a circumstance which can, as a rule, though not invariably, be attributed to an associated pleurisy with more or less effusion. Frequently a friction-rub is felt before complete consolidation is established.

*Percussion.*—Varying degrees of dulness are obtained in this stage, and before the lung-tissue becomes thoroughly solidified the note may have a tympanitic quality. After complete consolidation there is usually marked or absolute dulness posteriorly, while the note may be more or less tympanitic anteriorly, where the vibrations are more apt to reach the air in the larger bronchi. A sense of resistance is offered to the pleximeter-finger, but not to the same degree as in the case of a pleurisy with effusion. When the latter condition is associated the percussion-note will be flat. Deadness is less marked in old people in whose ribs senile changes have taken place, which render them more resonant, or in cases in which the consolidated areas occupy the central portions of the lung. Above the solidified part Skodaic resonance is usually obtainable.

*Auscultation.*—Bronchial or tubular breathing is heard, as a rule, over the solidified lung, but it may be absent in consequence of the plugging of the large bronchi with exudate (so-called *massive pneumonia*). Bronchophony is usually obtainable over the portion of the lung affected, though this may also be absent, and for the same reason as in the case of the bronchial breathing: it sometimes takes the form of egophony. Subcrepitant râles, due to associated bronchitis, are sometimes heard, and the crepitant râle at the end of inspiration, supposedly very characteristic, is best heard at the beginning of consolidation, when the pleura receives its coat of fibrin and while the lung is yet capable of sufficient movement to produce fine pleural friction. A distinct friction-rub may also be heard occasionally.

**Stage of Gray Hepatization.**—With beginning resolution the solid contents of the air-cells liquefy and are removed, so that air now re-enters the air-cells and permits a consequent increase in the movement of the lung.

*Inspection.*—The normal expansile movement of the affected side gradually returns.

*Palpation.*—Tactile fremitus progressively diminishes.



**Percussion.**—The dull or tympanitic quality of the note is gradually lost, though the fact must be emphasized that the abnormalities in the note vanish more slowly than the other abnormal physical signs. Some degree of deadness often remains long after recovery is apparently complete.

**Auscultation.**—With increased movement of the lung there may be a reappearance of the crepitant râle, due to interplay of the pleural surfaces, and the softened exudate in the air-cells gives rise to subcrepitant râles, heard both on inspiration and expiration, with coarser râles over the bronchi. Bronchial breathing gradually gives place to bronchovesicular, and the latter in turn to normal breathing.

**Complications.**—Doubtless many of these are due to the primary infection by diplococci.

**Pleurisy** is, of necessity, associated in all instances in which the consolidation reaches the pleura. It is to be looked upon as a direct result of the pneumonic process, since in a great proportion of cases examined the presence of the diplococci has been demonstrated. Cases are met with, however, in which the truly pneumonic symptoms are overshadowed by the intensity of the pleuritis, and to these the term *pleuro-pneumonia* has been applied. In this form there is often a copious effusion which is exceedingly rich in fibrin—a circumstance which distinguishes it from other forms of acute pleurisy. There may be the ordinary grade of pleurisy on the side affected by the pneumonia, and a severe grade on the opposite side, and when effusion occurs under the latter exceptional conditions it is apt to be purulent. Indeed, empyema has of late been shown to be a frequent complication of pneumonia, but, as far as my own observation goes, it would appear to rank as a sequel rather than a complication, coming on as it usually does several days after the crisis. Its development is accompanied by replacement of ordinary dulness by flatness with great resistance, and by the disappearance of râles and breath-sounds, normal and abnormal. Other characteristic features of empyema are present, but in the event of doubt surrounding the diagnosis the needle should be introduced.

There is a prompt rise of fever, the temperature leaping to 103° or 104° F. (40° C.) quickly, after which it is decidedly remittent in type, but there are no hectic chills. Fistulous connection with a bronchus, however, and the establishment of *empyema necessitatis* are common events in this form of the disease, and may be preceded by diurnal chills, sweats, etc.

The occurrence of septic phenomena is a certain indication of secondary infection by streptococci. The pus is absorbed very rarely, and more frequently becomes encysted. I saw one instance in which the effusion measured 8 liters, while ordinarily the amount ranges from 2 to 5 liters. Removal of the effusion by aspiration is promptly followed by the disappearance of the fever, but reaccumulation generally occurs, with another rise of temperature.

Finally, if defervescence takes place by lysis or if the “critical” decline is absent, a residual purulent or sero-fibrinous effusion may be considered as the likely cause. This latter complication is attended by a paroxysmal cough which is excited by movement, and is not usually accompanied by expectoration, while the temperature rises, though not



so high as when the effusion is purulent. It remains to be pointed out that rarely also there is a primary empyema, due to the pneumococcus.

**Acute general bronchitis** may pre-exist or may arise as a complication, and often proves formidable, intensifying the fever and increasing the dyspnea, the tendency to heart-failure, and the cyanosis. The expectoration of mucus is freer than in uncomplicated pneumonia, and over the bronchi moist râles intermingled with sibilant and sonorous râles are audible.

**Pericarditis.**—This is one of the most important complicating affections. It results from a direct extension of the adjacent pleuritis, and hence is more common in left- than in right-sided pneumonia, and children are more prone to it than adults. Although generally of the plastic variety, it is not infrequently sero-fibrinous, and rarely the effusion is purulent. The *diagnosis* can be made in the same way as when other conditions attend its development, but it may be readily overlooked by the careless observer. I would say, however, that the occurrence of increased dyspnea, with or without precordial pain, should serve as a warning and lead to a physical examination.

**Endocarditis.**—This is far more frequent than pericarditis, and particularly in the ulcerative form. Out of 209 cases of malignant endocarditis collected by Osler, 54 cases occurred in pneumonia, and my experience fully agrees with his statement as to its great relative frequency in this disease. There are no reliable symptomatic indications of this condition, and of those symptoms that do appear the physical signs are least trustworthy. Frequently murmurs are entirely absent; and, on the other hand, the presence of a murmur, even though it be loud and harsh, is by no means diagnostic of the condition. Some claim that a rough diastolic murmur is quite significant; this has not, however, been present in any of my own cases. The development of septic manifestations, especially irregular fever, chills, and sweats, renders the case highly suspicious, and when in addition there arises distinct evidence of embolic processes the diagnosis becomes highly probable. If, now, the symptoms of meningitis should supervene, little doubt would remain as to the character of the complications, since meningitis and endocarditis are often combined in pneumonia.

Netter, Weichselbaum, and Bignami have shown by microscopic examination and cultivation that acute endocarditis may be caused directly by the diplococcus of pneumonia, and, from the presence of this bacillus in the right ventricle, that it is far commoner than the forms due to other causes.<sup>1</sup>

**Chronic Endocarditis.**—This predisposes to acute endocarditis, both simple and ulcerative, but, independently of the acute form, pneumonia arising in the course of chronic endocarditis is apt to be attended by cardiac failure, with venous stasis as the consequence. The murmurs of chronic valvular disease often disappear with the development of pneumonia.

**Cardiac clots** (ante-mortem) may form, but are rare. They result from weakness of the ventricular wall, especially in the right heart; and are most apt to arise, therefore, in cases in which the death-agony is much prolonged. Venous thrombosis is rarely seen, and embolism of

<sup>1</sup> *Practitioner*, London, Aug., 1894.

the larger arteries is a rare complication. Cerebral embolism, causing aphasia and even hemiplegia, has been observed but seldom.

**Acute purulent meningitis** is a comparatively rare but very grave complication. It is often related etiologically to pneumonia, and its symptoms are not clearly defined; particularly is this true when it develops during the invasion-period and the basilar meninges are not involved. Hence its diagnosis is often impossible. The presence of intense and persistent headache, rigidity of the nucha, wild delirium, followed by stupor deepening into profound coma, affords a basis for a probable diagnosis. Its frequent association with ulcerative endocarditis and the symptoms of the latter have already been pointed out.

**Peripheral neuritis** is among the rare complications of this disease. Its presence is attested by the usual symptoms.

**Parotitis** is also sometimes seen, and may cause a fatal termination of the case. I have seen two instances, however, in which this was a complication, and both ended in recovery. It is thought to be associated usually with endocarditis, but in neither of my own cases were there any symptoms pointing to the latter affection.

**Arthritis.**—This may arise at any period in the course of pneumonia, and at the start may closely simulate rheumatism. It soon, however, takes the form of a purulent arthritis, and may be associated with other suppurative inflammations (meningitis, endocarditis). The pneumococci have been found in the affected articulation.<sup>1</sup>

**Gastro-intestinal Complications.**—*Croupous gastritis* may rarely intervene, but *croupous colitis* is a more frequent concomitant, giving rise to tympanites and marked diarrhea, and it may prove a serious condition.

*Peritonitis* occurs, but with great rarity.

**Jaundice** may be observed in all types of the affection, though, on the whole, it is more frequent in serious than in mild forms of the disease. Apart from the cutaneous and conjunctival discoloration, which is rarely intense, it has no symptoms in the majority of instances, and yet even in such it is most probably obstructive (hepatogenous). I have observed cases in which the evidence of a certain degree of obstruction was unmistakable, and these are to be ascribed to the presence of duodenal catarrh with extension into the ducts.

**Acute nephritis**, generally of a mild grade, is a rare sequel or complication, and its recognition is entirely dependent upon the discovery of albumin and casts in the urine.

**Clinical Varieties and Anomalous Types.**—(1) **Typhoid Pneumonia.**—This relates to an adynamic type of the disease with *typhoid symptoms*, and not to typhoid fever. It is often secondary to low fevers, to septicemia, diabetes, and chronic nephritis, and is also the variety met with in drunkards and in persons previously enfeebled on account of unhygienic surroundings. The onset is more gradual than in typical pneumonia. The physical signs may be well marked or ill defined, but in either event the general features are at once striking and characteristic. Prostration is extreme; there are delirium and often stupor; the temperature may or may not be high; while the respirations and pulse are almost always frequent. The skin is dry, and not infrequently there is a dusky tint or slight jaundice. The tongue is dry, often brown, and

<sup>1</sup> Bernheim, *La Médecine moderne*, Paris, Feb. 21, 1894.



vomiting is more common than in ordinary pneumonia; the sputa may be rusty or decidedly hemorrhagic. Splenic enlargement is often clearly perceptible. In this form of the disease there are numerous perils to pass, and when recovery ensues convalescence is long and tedious. This clinical form is not to be confounded with pneumo-typhoid, in which typhoid fever begins with pneumonia.

(2) **Epidemic Pneumonia.**—This is often of malignant type. The symptoms exhibit noticeable variations, according to the special etiology and to different epidemics. The pneumonias that have developed so frequently in the course of *epidemic influenza* have been complicated with or preceded by general bronchitis. The heart-power in many cases became exhausted early, and then followed congestion and edema of the lungs. The physical signs are often slight, even in fatal cases. In certain epidemic forms of pneumonia still other complications may be pronounced (cerebral, intestinal, etc.). In this connection should be mentioned so-called "*larval pneumonia*," in which the general symptoms are mild and the local signs ill defined. This sub-variety is observed in those epidemic outbreaks that occur in institutions, tenement-houses, jails, etc.

(3) **Latent Pneumonia.**—To this class belong *central pneumonias*, which have been described briefly under General Symptomatology. In these instances the sputum is to be stained and examined microscopically, when the pneumococcus will be found. When pneumonia arises in the course of emphysema a physical examination often gives negative results, and hence, the dilated air-cells not being filled with the fibrinous exudate, dulness is less marked than in typical pneumonia, and tubular breathing is often absent. The sputum is gummous and rusty, as a rule, and should be studied bacteriologically. Generally, but not always, before the crisis occurs consolidation advances to the periphery, when a physical examination will give positive results.

(4) **Migratory Pneumonia.**—By this is meant an extension of the specific inflammation to other parts of the lungs. This may occur at the time of the appearance of abundant moist râles in, and free expectoration from, the lobe first affected. Such extension prevents the occurrence of the usual crisis, and often occasions an exacerbation of the general pneumonic features.

(5) **Bilious Pneumonia ("Malarial Pneumonia").**—When lobar pneumonia occurs in persons who are subjects of malarial poisoning the initial chill is prolonged and the fever paroxysmal or decidedly remittent. Jaundice and vomiting are more common than in the ordinary type.

(6) In **children**, particularly in the very young, the first symptom is often a convulsion. Cerebral symptoms, as delirium, stupor, or even coma, may appear early. The upper lobes of the lungs are more frequently involved than in adults. Unless the objective indications be examined for, the disease is frequently overlooked. The characteristic sputum is rarely seen in juvenile pneumonia.

(7) In **old persons** pneumonia runs a peculiar course and is dangerous in the extreme. Most cases begin less abruptly than in younger persons, the initial chill being often absent or replaced by moments of chilliness. There may be nausea and vomiting, and anorexia is usual. Prostration sets in early and is profound, and there is fever,



but this does not range so high, and its type is more irregular than in non-senile pneumonia. Nervous phenomena, sometimes prominent, are not uncommon, but the local symptoms (cough, expectoration, and pain) are feebly developed or wholly absent. The area of lung-tissue implicated is often insignificant, the physical signs being slight or even entirely wanting; and when present there is usually dulness on percussion (with a tympanitic quality), tubular breathing, and a few subcrepitant râles. The physical signs are often sharply localized and their character variable. This affection, as it occurs in old people, is a most deceptive one, the cases very generally ending fatally after an illness of an apparently mild degree of intensity.

**Relapses.**—These are among the rarest of events, and are usually rudimentary when they occur. *Recurrences* are ordinary, however, second, third, fourth, and even more attacks having been noted in the same individual (*vide* Etiology).

**Course and Duration.**—In cases which recover the febrile stage lasts from three to thirty or more days. In most instances, however, defervescence occurs on the fifth or seventh day, and resolution is completed about one week later, making the total duration from twelve days to two or three weeks. Convalescence may be delayed when complications outlast the primary disease or when sequelæ arise, and fatal cases are most apt to terminate on the seventh, eighth, and tenth days of the disease. It remains to be added that the regular course of pneumonia is often greatly modified by the various complications (endocarditis, pericarditis, meningitis, etc.).

**Termination.**—(a) **Delayed Resolution.**—The process of resolution, consisting in softening of the exudate and its subsequent removal (partly by absorption, partly by expectoration), may not begin until the fourth, sixth, eighth, or even tenth week. Usually defervescence with a moderation of the other general features has taken place long before the physical signs indicate resolution. When the latter change occurs it may lead to complete restoration of the functional and anatomic entirety of the lung-tissue, or proliferation of the interstitial connective tissue may arise during the period of postponed resolution, producing (b) **chronic interstitial pneumonia**. This is very rare, however (*vide supra*, Pathology). (c) **Abscess** and (d) **gangrene** are also rare sequelæ. For their clinical description the reader is referred to the description of Diseases of the Lung.

**Diagnosis.**—The diagnosis is determined by special local and general symptoms, together with the physical signs. Of these, the abrupt onset with rigor, the course of the fever with termination by crisis, the stabbing chest-pains, the dyspnea, the peculiar type of breathing, the abnormal pulse-respiration ratio, the cough, the rusty expectoration, and the signs of consolidation of one or more lobes of the lungs, are the most characteristic. Deviations from the usual type are frequent, and these do not present many of the distinctive features just mentioned. Among the latter the so-called “*typhoid pneumonia*” and the other clinical varieties whose most valuable diagnostic features have been given, are often difficult of recognition. It must not be forgotten that repeated physical examinations of the chest will often detect more or less extensive consolidation, even though local symptoms are entirely

wanting. Again, when in the course of certain chronic affections (phthisis, cancer, Bright's disease, diabetes, and organic affections of the heart) more than the customary degree of fever is developed, physical exploration of the heart and lungs is imperatively demanded. Be it remembered that in this class of cases "the physical signs are obscured, because respiratory action is enfeebled by the primary condition" (Musser).

**Differential Diagnosis.**—This relates to (a) acute pneumonic phthisis, (b) typhoid pneumonia, (c) meningitis, (d) broncho-pneumonia, (e) acute pleurisy with effusion.



FIG. 17.—Lobar pneumonia: 1, unaffected area (upper lobe); 2, consolidated area (middle lobe); 3, resolving area (lower lobe); 4, heart in normal position.

(a) PRIMARY LOBAR PNEUMONIA.

There may have been prior attacks.

Sudden, with severe rigor and rapid rise of temperature.

Fever of continued type, terminating by crisis.

No drenching sweats, except at time of crisis.

ACUTE PNEUMONIC PHTHISIS.

Inherited predisposition or previous tuberculous disease.

Generally more gradual—repeated fits of chilliness (rarely severe rigor), often following exposure or "cold."

Fever of remittent type, often becoming intermittent, without crisis.

Drenching sweats present and oft repeated.

Herpes common.  
Not much emaciation.  
Pulse-respiration ratio considerably disturbed.

Sputum rusty-colored, viscid, and sticky ; contains pneumococcus.

Duration of febrile stage shorter.

Physical signs, as a rule, first referable to base of lung.

Absent.

Rapid emaciation.

Less so.

Sputum may be blood-tinged ; is more purulent and more copious, and contains numerous bacilli and yellow elastic tissue.

Duration longer.

First referable to apex.

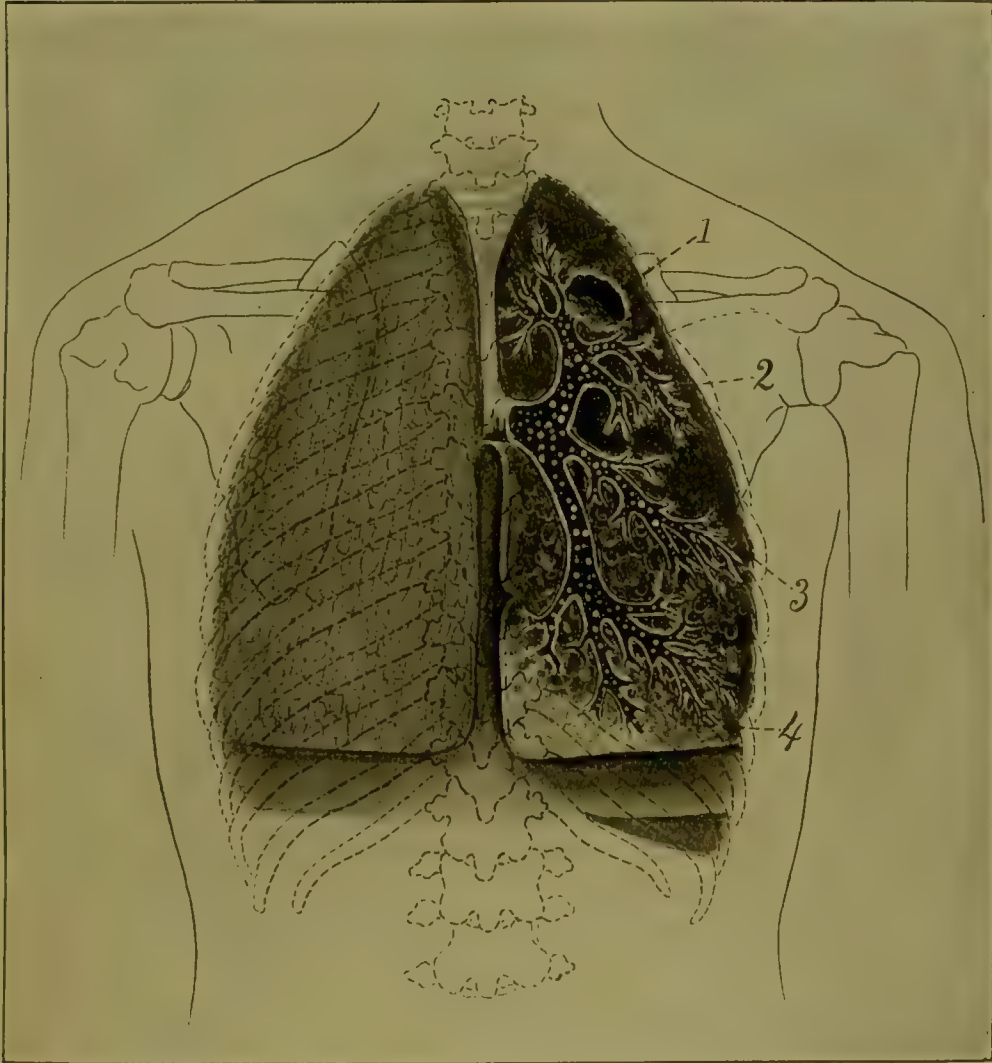


FIG. 18.—Acute pneumonic phthisis, posterior view : 1, cavity ; 2 and 3, consolidation ; 4, infiltration ; the white spots indicate râles.

Usually limited to one lobe or the lower portion of one lung.  
Signs of consolidation, followed by resolution.

Apex of healthy side not involved.

Prognosis not hopeless.

Tuberculous disease of other organs does not follow as a rule.

Usually extension from apex to base.

Signs of consolidation, followed by cavity-formation, with large gurgling râles at apex.

Apex of opposite side generally invaded.

Hopeless.

Often does.



(b) **Typhoid pneumonia** must be diagnosed from pneumo-typhoid, and the blood in the two conditions may be of service in the discrimination. Leukocytosis usually exists in pneumonia, and there is hypoleukocytosis in typhoid; but this fact is only of value when there is marked increase or decrease of the leukocytes, since figures about normal may occur in either condition.

Widal's test will be a decided aid. His assertion that a drop of blood from a patient with typhoid, added to a pure culture of typhoid bacilli, causes the cessation of the motion of the bacilli and their collection in clumps, and that this does not occur with blood from other diseases, is, I think, satisfactorily proved.

(c) **Meningitis** is sometimes mistaken for pneumonia, and particularly when the latter occurs in children. The initial symptom of pneumonia in the very young is often a convulsion; whereas, though in meningitis this form is not uncommon, it is more apt to manifest itself later. When headache occurs in pneumonia it is frontal. It is almost invariably complained of in meningitis, but is occipital, and is associated with rigidity of the cervical muscles. Before the occurrence of pressure-symptoms in the latter disease the patient is very restless and morose; his reflexes are exaggerated and there is marked hyperesthesia. The temperature-range is lower, more irregular, and there is no crisis, while the pulse is more variable and often irregular in meningitis. In pneumonia with latent local symptoms the pulse-respiration ratio is greatly altered and the type of respiration peculiar (*vide ante*). The important rule, to examine for the physical signs in doubtful cases, must not be neglected, and if the subject be young the apex region in particular.

The differential diagnosis between pneumonia and broncho-pneumonia and pleurisy with effusion will be found on pages 517 and 551.

**Prognosis.**—The mortality from pneumonia in hospitals averages about 25 per cent. It is less in private practice—about 15 per cent. The death-rate, however, is greatly modified by the type of the individual epidemic, and by so many conditions and incidents that a precise statement as to the percentage of fatal cases cannot be ventured. The above mortality-rates have been based upon all of the accessible statistics at my command. Wills collected 223,730 cases, which gave a mortality of 18.1 per cent.

The elements that enter into a correct prognosis are in the main identical with those in other acute infectious diseases, and concern (1) the severity of the type of infection, (2) the presence or absence of complications, and (3) circumstances peculiar to the individual.

(1) **Severity of the Type of Infection.**—In asthenic cases this is shown by (a) the temperature-range, (b) the degree of heart-power, (c) the intensity of the nervous symptoms, and to some extent by (d) the size of the area of lung-induration. It has been demonstrated, experimentally, that the absence of leukocytosis is indicative of a grave type.<sup>1</sup> In case the diplococcus be found in the blood, the prognosis must be considered very grave, as it has never been found there during life, except in cases that are in themselves very grave or seriously complicated. A continuance of marked leukocytosis with a drop in temperature would point to a pseudo-crisis. (a) **The Temperature-range.**—A continued high tem-

<sup>1</sup> Von Jaksch and Tschistowitsch, *Annual of the Universal Med. Sciences*, vol. i., 1893.

perature, as, for example, 105° F. (40.5° C.), on two or three consecutive days without material remissions, is ominous. (b) **The Degree of Heart-power.**—A steadily rising pulse-rate after the fifth day indicates real danger, since it points indisputably to gradual cardiac failure. The same thing is shown by a diminution in the intensity of the second pulmonary sound, particularly the giving out of the right ventricle. (c) **The Intensity of the Nervous Symptoms.**—Active delirium is not favorable at any stage, and is particularly unfavorable if it develop early. When it assumes the form of delirium tremens the case has usually passed beyond hope of recovery. (d) **The Size of the Area of Lung-induration.**—I have observed that extension of the consolidation at an advanced stage belongs to serious types. The same may be said of double lobar pneumonias.

**Typhoid pneumonia**, being of asthenic type, gives an unfavorable prognosis, notwithstanding an absence of high temperature and of extensive inflammation of the lung-tissue.

(2) **Presence or Absence of Complications.**—Cases in which there is involvement of a single lobe or two lobes, if it occur on the right side and without complications, generally terminate in recovery. In nearly one-half of the instances complications occur, and these greatly increase the death-rate. Among the most common is *pleurisy*, which, unless accompanied by considerable effusion, does not add fresh danger; when pleurisy attacks the unaffected side, however, it does. *Empyema*, following pneumonia, generally terminates in recovery unless secondary septic phenomena appear. Extensive *bronchitis* is a most perilous complication in my judgment. *Pericarditis* decreases the chances for recovery, but by no means to the same extent as *endocarditis*. *Cardiac clots* may form, but usually the patient is already moribund. *Abscess of the lung* and *gangrene* form highly unfavorable complications. *Con-gestion* and *edema* of the uninvaded portions of the lungs render the outlook bad, and these, together with cyanosis, are apt to be dependent upon failure of the right heart. *Acute meningitis* is exceedingly grave. Fenwick, as the result of an analysis of 10,000 cases, found that the quantity of albumin in the urine is of considerable prognostic value. *Gastro-intestinal* complications occurring at the outset are unpropitious.

(3) **Circumstances connected with the Individual.**—Of these *age* heads the list, and after the twentieth year the mortality increases progressively until the seventh decade. It has been claimed that nine-tenths of the deaths after the seventy-fifth year are from lobar pneumonia. Under the twentieth year, according to the analysis of 708 cases at St. Thomas's Hospital by Hadden, H. W. G. Mackenzie, and W. W. Ord, the mortality is 3.7 per cent., while in infancy it exceeds that of early childhood.

**Sex** has little influence, though the disease is believed by some to be more fatal in females than in males. In the *debilitated* the danger is greater than in the vigorous, and the *alcoholic* rarely escapes death.

**Modes of Death.**—Most frequently death is immediately due to heart-failure, which results from two causes: (1) overwork, as when an extensive area of lung-tissue is involved; and (2) the direct effect of the pneumotoxin upon the heart. The *complications* mentioned may prove fatal, however, and in one of my own cases thrombosis of the coronary



artery killed the patient. This may be a not uncommon terminal condition.

**Treatment.**—**General Management.**—The patient should occupy a well-aired apartment, which should be maintained at a temperature of 65° F. (18.3° C.), except in pneumonias occurring in the very young, when it should be several degrees higher. The patient should not be allowed to leave his bed for at least one week after the occurrence of the crisis; and as pneumonia is a self-limited affection, the principal object is to support the powers of life until the crisis is passed. *To this end nothing contributes so much as proper feeding.*

The **diet** should be light, chiefly liquid, but of the most nutritious sort. Alimentation should be especially vigorous when there is the slightest tendency to increasing debility. On the other hand, in uncomplicated cases and in those in which the disease is limited to a single lobe there is not the same need of supporting the vital powers, since these cases have an intrinsic tendency to recovery. *Milk* should constitute the chief article of diet; meat-broths or meat-juices, egg-white, and light farinaceous substances may also be allowed. The food, and particularly the milk, is to be administered at stated brief intervals and in definite quantities. When resolution is delayed stronger forms of nourishment (scraped meat, etc.) may be given. After the crisis a gradual return may be made to the usual forms of solid foods.

**Cardiac stimulants** are often indicated. It is well to begin their use as soon as the slightest tendency to cardiac failure is shown. The evidences of the latter conditions are to be found in the state of the pulse, the first sound of the heart, and the pulmonic second sound. The pulse becomes more and more accelerated and feeble, the first sound of the heart less distinct on auscultation, and the pulmonic second sound loses its accentuated character. From the first moment that these unfavorable features or marked nervous symptoms appear alcoholics must be used. At first they are to be employed in moderate doses ( $\frac{1}{2}$  ounce—16.0—of whiskey or brandy every three hours), to be increased if the favorable effect be proportionate with the urgency of the indication. There is a great tolerance of alcohol in this disease, and in the pneumonia of drunkards its early and free use is to be recommended.

If the alcoholic stimulants fail to meet the above indications, other cardiac stimulants must be administered simultaneously. Of these, strychnin has been the most serviceable in my own hands, its mode of administration following the same rules as have been mentioned for alcoholics—at first in moderate-sized doses, to be increased as occasion demands. Should urgent need of stimulation arise, however, either suddenly or more gradually, strychnin should be exhibited hypodermically. It is my custom in desperate cases to use subcutaneously as much as gr.  $\frac{1}{15}$  (0.0043) every two or three hours. So soon as the condition of the heart denotes restoration of cardiac power the size of the dose is to be reduced, but the agent is not to be withdrawn until the disease has run its course. In no other disease does strychnin possess greater potency for good than in pneumonia, if wisely employed. For sudden heart-failure ether, administered hypodermically, is also very efficacious, and digitalis may be combined with the strychnin or given separately. Like strychnin, digitalis may become a life-saving drug if its adminis-



tration be guided by sufficient judgment. Ten minims (0.666) of the tincture may be given every three or four hours internally or hypodermically if needful, and I have found that the association of a small dose of nitroglycerin ( $m\frac{1}{2}$ ; 0.033, every three or four hours) considerably aids the action of the digitalis. Nitroglycerin is especially indicated when the renal secretion is scanty and the urine contains more than the usual trace of albumin. Ammonium, in the form of the aromatic spirits or the carbonate, is an excellent stimulant to the feeble heart of pneumonia.

**Respiratory Stimulants.**—Beginning cyanosis is the signal for the use of respiratory stimulants, of which the best are oxygen by inhalation, strychnin, and atropin. The oxygen must be administered in large amounts, and if the patient be so severely ill that almost constant inhalation of the gas becomes exhausting, it may merely be allowed to escape near his nose and mouth.

**Hydrotherapy.**—This is especially useful, but I have abandoned the rigid cold-bath method. The gradually-cooled tub-baths are the best, and should be employed, except in cases pursuing a very favorable course or those in which little besides rest and good nursing is necessary. In meeting high temperature, marked nervous symptoms, dyspnea, cardiac weakness, etc. they offer many superior advantages, and in pneumonia the effect of the baths upon the cardiac, respiratory, and nervous centers is especially desirable. It is of importance that the patient while being immersed put forth no muscular effort. He must be held and supported while in the bath, and gentle friction to the skin-surface must be made. The temperature of the baths should not be too low at the start: at the beginning it should be 90° F. (32.2° C.), and then be lowered according to the degree of sensitiveness of the individual patient. It is rarely necessary to go below 80° F. (26.6° C.), and in the aged, the very young, and in persons previously debilitated it is unwise to use any other than tepid baths. The duration should not exceed ten to fifteen minutes on the average, and more than three—or at most four—baths daily are not required. Cool sponging, combined with the ice-cap or the wet pack, may serve as a substitute when full baths cannot be employed.

**Abortive Method of Treatment.**—Petresco has found that large doses of digitalis (3j-ij; 8.0, of the digitalis-leaves in an infusion daily) administered at the onset will jugulate the disease. His experience covered 1192 cases, and showed the surprisingly low mortality-range of 1.22 to 2.66 per cent. This plan of treatment is rational, since it aims at meeting the chief pathogenic indication of pneumonia by passing through the lung-tissue an adequate proportion of leukocytes, and thus re-establishing the cardio-pulmonary circulation. In the few instances in which I have adopted the plan it has failed to cut short the disease, though the cases terminated favorably. After the full development of consolidation I would urge caution in the use of digitalis unless cardiac failure threaten.

**Venesection.**—It has been claimed by some that free bleedings at the onset will abridge the disease. Doubtless it is a good measure in sthenic cases (which occur with relative rarity in cities, but are not uncommon in rural districts), the temperature falling, the pain, the

dyspnea, and the nervous symptoms being relieved and the pulse softened. Later in the course of pneumonia venesection is to be resorted to if cyanosis and the signs of collateral pulmonary edema—due to a failing right heart—arise, and if cardiac and respiratory stimulants have proved futile. At this stage, however, bleedings rarely yield good results, though affording temporary relief.

**Antiseptic Methods.**—This aims to destroy the pneumococcus or, at all events, to neutralize the poisonous products of the latter in the blood, thus moderating the general disturbances. The method is based upon etiologic indications, and is most rational. The best antiseptics are carbolic acid (℥j ; 0.066, every four hours), thymol (gr. ij–iij ; 0.129–0.194, every four hours), mercuric chlorid (gr.  $\frac{1}{100}$  ; 0.0006, every four hours).

**Treatment of Special Symptoms.**—The initial **pain**, which is of an acute, agonizing character, is relieved by the hypodermic use of morphin at intervals of six or eight hours. This counteracts the shock produced by the invasion-period, but it is to be omitted if the bronchi contain secretory products, since morphin dries these and favors their accumulation rather than their removal. Rarely is it necessary to continue this remedy after the second or third day of the illness.

**Fever.**—The fever of pneumonia is a temporary affair, and instead of being hurtful may prove beneficial, since it furthers tissue-metabolism, and this aids in the destruction of the specific poison of the disease. Fortunately, internal antipyretics for the purpose of combating high temperature are not so largely used at the present day as formerly. It is true that they possess the power to reduce temperature, but their use is attended with danger from their action as cardiac depressants; while, if it be true, as before stated, that pneumonia usually kills through the heart, it follows that cardiac power must primarily be conserved. Apart from the above-mentioned serious objection to internal antipyretics, it is to be remembered that they do not possess the important additional advantages to be derived from cool baths. In my opinion, their use should be limited to those cases in which cool baths or their substitutes (cold pack and cold spongings) fail to effect a reduction of fever. It must not be forgotten that unless the temperature exceed 104° F. (40° C.) it had better be let alone. When called for, however, the best among the antipyretics are acetanilid and phenacetin. The dose of these should be small—gr. v (0.324) of the latter and gr. ij–iij (0.129–0.194) of the former, to be repeated at intervals of four to six hours if required.

Like internal antipyretics, arterial sedatives are to be used sparingly, and when used their effects must be carefully noted. In cases in which venesection is indicated the tinctures of veratrum viride and of aconite have been much vaunted as substitutes. The tincture of veratrum viride produces a good effect upon the local condition, since it relaxes the arterial walls, and thus bleeds the patient into his own vessels, but, since it also acts as a cardiac depressant, it is questionable whether its disadvantages do not outweigh its advantages. The tincture of aconite, owing to its depressing influence upon the heart, should not be employed.

The **nervous symptoms** are successfully met, as a rule, by hydrotherapy (including the ice-cap), by the arterial stimulants, and by the use of morphin, as before recommended.



**Cough** during the early stage is controlled by the morphin needed to combat the pain. In the more advanced stages, if there be present numerous moist râles and a scanty expectoration, stimulant expectorants (ammonium muriate, terebene) may be employed with happy effect; but ordinarily they do harm rather than good. Pilocarpin may aid resolution when this is delayed (Reiss); the heart, however, must be carefully watched over.

**Complications.**—The management of the complications does not differ from that which is appropriate when they occur as independent affections, though all depressing measures must be positively omitted. I would add that in pleuro-pneumonia aspiration is not well borne, according to my observations; hence, unless urgently needed, I would postpone this procedure until the crisis has passed.

**Local Measures.**—When in doubt as to whether venesection should be employed or not, it must be remembered that early local bloodletting (cupping and leeching) is followed by relief from pain and dyspnea, but that these measures should be reserved only for robust persons. Counter-irritation by means of sinapisms is useful at the onset. The cotton jacket has certain advantages in maintaining the free, local action of the skin, and may be employed; before the days of hydrotherapy it was quite commonly used. The *topical use of cold* in the form of ice-bags has been practiced extensively by Lees of England and Mays of America with brilliant success, cold thus applied relieving the pain and dyspnea and diminishing pyrexia. In my limited experience with the application of cold I have found cases in which it could not be tolerated by the patient, and in such, warm applications (poultices, etc.) exerted a soothing revulsive effect. Lépine has used with success very dilute mercuric chlorid injections into the affected lung-tissue. This mode of treatment has not been followed in sufficient cases to warrant an opinion as to the extent of its usefulness.

## SECONDARY PNEUMONIA.

**Pathology.**—The lesions are identical in character with those of primary lobar pneumonia, but the areas involved have not always the same regular distribution. Congestion surrounding the hepatized lung-tissue is not infrequently extensive. We see, post-mortem, a tendency to commingling with small areas of lobular pneumonia. Both the streptococcus and the micrococcus lanceolatus are frequently found on microscopic examination.

**Etiology.**—Most instances are secondary to the acute infectious diseases, and it is probable that the specific causes of certain of the latter (Eberth's bacillus, Pfeiffer's bacillus, etc.) have the power to excite the morbid changes of acute lobar pneumonia. *Colon-pneumonia*, due to the bacillus coli, is the result of hematogenous infection either from the intestinal or from the urogenital tract. In the majority of instances, however, in which this disease develops in the course of the acute infectious diseases the latter are to be regarded as merely furnishing the opportunity for infection by the micrococcus lanceolatus.

**Symptoms.**—The rational symptoms are often absent. Close observation may, however, detect more or less dyspnea, cough, and in-



creased fever, and rarely the attack is heralded by a rigor, followed by fever, the pneumonic type of breathing, pain, cough, and the characteristic expectoration.

The physical signs, when carefully observed, usually serve to enlighten the physician as to the nature of the affection. Hence it is a natural corollary that repeated physical examination is demanded in all cases in which there is danger of intervening lobar pneumonia.

**Diagnosis.**—This rests chiefly upon the physical signs, which are the same as in primary lobar pneumonia. Obviously, when the local subjective symptoms and the characteristic sputa are present a correct diagnosis is easily made. The fact must be emphasized that bronchopneumonia arises in the course of infectious diseases far more frequently than does lobar pneumonia.

**Prognosis.**—The occurrence of lobar pneumonia as an intercurrent affection adds greatly to the gravity of the primary disease. It is especially dangerous when it appears as a sequel during convalescence from acute infectious diseases.

**The treatment** is similar to that of primary lobar pneumonia, though less satisfactory.

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## INFLUENZA.

(*La Grippe; Epidemic Catarrhal Fever.*)

**Definition.**—Influenza is an acute contagious disease, caused by the bacillus of Pfeiffer. Its chief symptoms are due to catarrh of the respiratory and digestive tracts, together with profound muscular and nervous prostration, and grave complications (especially pneumonia) often present themselves. The disease may be endemic, though more often it is epidemic or pandemic.

**Historic Note.**—Every quarter of the globe has been the scene of visitations of epidemic influenza. More rapidly than any other disease belonging to the same class does it traverse a region of country, and hence within a remarkably brief period of time a whole nation may suffer. As a rule, influenza develops into epidemic proportions in the East, whence it spreads with unparalleled rapidity in a westerly direction. The first epidemic of the disease in the United States appeared in 1647, and was subsequently described; and, though it has since then frequently prevailed, the outbreaks have not observed any regular periodicity. The last true pandemic of the affection originated in Bokhara in May, 1889, reached St. Petersburg in the following October, Paris in November, and London in turn early in December. In America the cases began to appear about the middle of December, and rapidly multiplied into an explosive epidemic, which reached its maximum in January, 1890. Influenza reappeared in epidemic form, though less extensively, during April and a part of May, 1891, and again in a briefer and lighter form in the winter of 1891–92. During the winter of 1892–93 only a few sporadic cases occurred. In the early part of

1895 the disease assumed epidemic prevalence, and it again appeared at the beginning of the present year (1897).

**Pathology.**—There are no special anatomic lesions that characterize the disease. The rare instances in which death occurs in uncomplicated cases simply show marked catarrhal implication of the respiratory, and usually also of the gastro-intestinal, mucosa. There are practically no changes in the glands of the digestive mucosa, except in the abdominal type of the affection, in which there may be enlargement of the glands of Peyer and of the solitary follicles. As will be seen hereafter, most of the fatal cases exhibit lesions which are to be ascribed to the complications. Among the latter are pneumonia (either lobular or lobar), with which plastic pleurisy is usually associated, sero-fibrinous pleurisy, empyema, purulent pericarditis, nephritis, and rarely cerebro-spinal meningitis.

**Etiology.**—That the disease is microbic in origin can no longer be doubted.

**Bacteriology.**—Early in the year 1892, Pfeiffer discovered a bacillus which he has shown to be the true cause of influenza—the *bacillus of Pfeiffer*. It is of about the same breadth as the bacillus of mouse-septicemia, and only one-half the length of the latter. When stained with Ziehl's carbol-fuchsin it may be observed as a small dumb-bell, having knobbed ends connected by a rod-like shaft. These bacilli are obtained from the sputum, and their number bears a definite relationship to the intensity of the disease. They are found only during the attack, and are never present in any other disease. Pfeiffer has shown that they may penetrate the peribronchial tissue and pass out to the pleura. They have also been found in the blood. This bacillus can be cultivated in agar and other media, but not in gelatin, and when inoculated into rabbits and other animals it causes more or less typical influenza; these experiments, however, are not in themselves conclusive.

**Modes of Conveyance.**—Naturally, a specific germ that is propagated with the unusual rapidity that marks the bacillus of Pfeiffer must be air-borne. Even this view, however, fails to explain satisfactorily the coincidental prevalence of the malady at widely-separated points. Pepper suggests that the micro-organism may be almost universally distributed, but only capable under ordinary circumstances of causing occasional and sporadic cases; and that under certain extraordinary atmospheric or telluric conditions it acquires a degree of virulence that renders all subject to its attack. There is no doubt that influenza is communicable by *contagion*, and evidence is not wanting even to show that it may be transferred by fomites.

**Manner of Invasion.**—How the contagion enters the system has not been positively determined, though it is probably with the inspired air through the respiratory tract. Some authors contend that it may enter through the alimentary canal, while still others believe that the primary point of infection is not rarely the conjunctiva.

**Predisposing Causes.**—These are few and unimportant, since all persons are liable to the contagion. *Age* has slight influence, the period of greatest susceptibility being from the twentieth to the thirtieth year. The very young are less liable than older subjects, and during an epidemic are apt to be affected last, while old persons (particularly if debil-



itated) are frequent sufferers. The same is true of those whose vitality is lowered by neuropathic heredity or chronic maladies, these being among the first to be affected during an epidemic. On the other hand, it is to be remembered that the healthiest are not exempt. The affection is only slightly influenced, if at all, by meteorologic conditions.

**Immunity.**—A primary attack of influenza does not bestow immunity, since relapses are very common, and sometimes after long intervals. Many persons, too, suffer from the disease with the reappearance of fresh epidemics, so that two, three, four, or even more attacks may be observed in the same individual.

**Antagonism.**—Recent investigations have shown that a decided antagonism exists between influenza and malaria,<sup>1</sup> and that during epidemic prevalence of the former the latter has repeatedly suffered a great decline.

**Clinical History.**—**General Symptomatology and Course.**—The incubation period is quite brief, rarely exceeding two or three days. The onset is generally sudden, with either a severe rigor or repeated slight shiverings, accompanied by a rapid elevation of temperature which may touch 104° or 105° F. (40.5° C.), intense headache, distressing myalgic pains, and great prostration. The primary fever, however, varies greatly in severity. The same is true of the character of the symptoms—both local and general—presented by different cases as well as by different epidemics. Profound prostration characterizes the vast majority of instances during the invasion period. Depression of spirits, restlessness, insomnia (more rarely undue somnolence), and frequently delirium, are among the more prominent nervous phenomena. In certain epidemics the affection may be ushered in by vertigo—a symptom that sometimes also appears late in the disease.

The most striking symptom is *pain*, which in a certain percentage of cases is referable chiefly to the forehead, temples, occiput, eyeballs, and root of the nose. General neuro-muscular pains, however, are apt to be present. Their principal seat is often in the region of the lumbar spine (rachialgia), whence they are apt to dart upward to the neck and downward to the lower extremities. With the universal myalgic pains there is a general soreness, and I have frequently noted cutaneous hyperesthesia. The pains take the form of neuralgia of individual nerves or of pleurodynic stitches, or there are localized areas of burning, boring muscular pain. The *temperature* may, as before intimated, mount quite high at the beginning, and if so it usually remits during the first night. It subsequently remains at a comparatively low point, with evening exacerbations, until the normal level is reached, which often occurs as early as the second or third day, but may be postponed until the end of a week. The temperature-curve is markedly irregular, and often terminates by an apparent crisis. The *pulse* is small, feeble, irregular, and even intermittent, and I have sometimes observed it to be unusually slow, cardiac debility being prominent and at times reaching a dangerous degree. In many cases *dyspnea* is a rather conspicuous

<sup>1</sup> "A Statistical Study of Influenza; its Potency to Lessen the Receptivity of the Body for Malaria, as well as to Increase the Receptivity for Pneumonia and, probably, Typhoid Fever," by the author—*Philadelphia Hospital Report*, 1895, vol. iv.



symptom, occurring independently of inflammatory pulmonary complications. The same is true of *cyanosis*.

**Clinical Types.**—Different types have been described based on the differences in the local manifestations. But it is to be mentioned that influenza is remarkably protean in its clinical features, and that the enumerated types quickly and frequently merge into one another. (a) *Respiratory Type*.—Local catarrhal symptoms usually develop in the course of one or two days. They are, as a rule, evidenced first by a suffusion of the conjunctivæ, with excessive lacrymation, frequent sneezing, and slight pharyngitis. A little later, in most instances, hoarseness and cough come on, the latter being hard, racking, paroxysmal in character, and resembling whooping-cough. The cough and other local symptoms are due to intense, dry laryngo-tracheal irritation. In most instances the expectoration is scanty, and in these the physical signs are very generally negative. In a smaller proportion of the cases there is considerable expectoration, and the physical signs of ordinary bronchitis are manifested. (b) *Gastro-intestinal Type*.—The catarrhal symptoms sometimes center in the digestive system, and most frequently in children. In such, vomiting comes on early and is apt to be repeated at longer or shorter intervals. There is diarrhea, more or less urgent, with sharp abdominal pain, as a rule. (c) The *cardiac* group of symptoms that occasionally supervenes comprises heart-failure and distress, with a rapid, feeble pulse. (d) *The typhoid type* presents a continued fever, with the signs of the typhoid state. Nervous symptoms are very marked, such as stupor or delirium, dry, brown tongue, etc. (e) *The rheumatoid type* manifests itself by violent pains in the muscles all over the body. There is no visible change in either the joints or the nerve-trunks.

After the temperature has become normal profound prostration is apt to continue, and hence the patient shows no disposition to muscular or mental exertion. In cases of average severity convalescence is usually somewhat protracted, and in the severer forms decidedly so.

**Complications.**—(1) **Pulmonary.**—An ordinary bronchial catarrh may be properly regarded as belonging to the peculiar processes of the disease, but severe bronchitis, particularly affecting the capillary tubes and leading to broncho-pneumonia, is a common and very serious complication. As a secondary result we are apt to observe the development of collateral pulmonary edema, with its usual fatal termination; and, whilst this complication is prone to develop in the so-called thoracic type of influenza, it is by no means limited to this class of cases. In the latter it originates apparently in the profound prostration of the nervous system—a condition which also annuls in great part the phagocytic action of the leukocytes. In nearly all instances, however, this form, as well as croupous pneumonia, may be definitely traced to exposure. The bronchial glands may become acutely enlarged.

*Lobar pneumonia* is also a frequent and very fatal complication. It may arise early and in rare instances insidiously, but it is much more apt to manifest itself after influenza has about exhausted its force upon the vital organs or during the early part of convalescence. The symptoms of invasion—severe chill, high temperature, followed by the usual physical signs—are sudden in their onset and lead rapidly to an extremely serious condition.

When lobar pneumonia develops early in the course of influenza (a rare event), its symptoms are modified, the preliminary chill and pain in the side being often absent, and more frequently still the characteristic crepitant râle. Subcrepitant râles, however, are audible, and the dyspnea is out of proportion to the area of lung-tissue involved. Most of the peculiar features just pointed out may also be observed in connection with the pneumonia that appears during convalescence, though, according to my own experience, they are then more feebly expressed.

The recognition of *broncho-pneumonia* is in many instances quite difficult. Here, as elsewhere, it is secondary to general bronchitis affecting the larger tubes. I have observed this condition in cases in which the physical signs of bronchitis were not presented prior to its onset. In other instances, however, a few scattered sibilant and sonorous râles, intermingled with a few moist ones—the usual auscultatory signs of severe general bronchitis—are noted, and then broncho-pneumonia supervenes. Immediately preceding the signs of consolidation in broncho-pneumonia (less frequently also in croupous pneumonia) the respiratory murmur becomes exceedingly weak, and later an abundance of subcrepitant râles becomes audible over the affected area. Bronchial breathing may be associated, though it is rarely marked; but when the spots involved are of considerable size, they may give rise to corresponding zones of dulness on percussion.

According to some authors, the nature of the condition is variable, and the symptoms that simulate more or less closely those just depicted are ascribed to *congestive collapse* and other conditions, rather than to the ordinary type of broncho-pneumonia. *Congestion associated with edema* of the lungs occurs as a complication of influenza, as I have learned from personal observation. Acute enlargement of the bronchial glands may also be noted, and the recognition of this condition may be aided by careful percussion over the upper four dorsal vertebræ, where dulness will be obtained.

**Plastic pleurisy** is commonly an associated condition, especially in cases of lobular or lobar pneumonia. Other forms of pleurisy also occur, though less frequently (sero-fibrinous and empyema). *Gangrene* and *abscess* of the lungs may arise as terminal complications.

**Cardiac Complications.**—Heart-failure often manifests itself, and may prove fatal, though rarely. Purulent pericarditis is a rare complication, and is often secondary to pleurisy or pneumonia, while attacks of angina, which usually interchange with simple weak heart (often associated with arrhythmia), have been noted in certain epidemics (Curtin and Watson).

**Gastro-intestinal System.**—There may be severe gastro-enteritis, with frequent vomiting and purging and intense abdominal pains, and, more rarely, hemorrhages occur from the stomach and bowel. Catarrhal jaundice may appear, due to duodenal catarrh, but these gastro-intestinal complications are more apt to be met with in young children than among adults.

**Nervous System.**—The most frequent symptom is perineuritis, which probably causes much of the patient's sufferings. Delirium of a most active form sometimes appears, and particularly when certain other complications have arisen, such as pneumonia, pericarditis, etc. Cerebro-spinal meningitis occurs as a rare complication, and when it arises



is to be attributed to secondary infection with the streptococcus. I have observed symptoms identical with those of meningitis appearing suddenly, and in the course of a day or two disappearing just as suddenly. The symptoms under such circumstances must be due either to the action of the specific poison upon the nerve-centers or to congestion, and hence a diagnosis of suppurative meningitis is to be made with extreme caution. In addition to the presence of the symptoms which characterize the affection, we should have the existence of suppuration elsewhere in the body (otitis, purulent pericarditis, etc.) or the presence of pneumonia. Cerebral abscesses have also been noted (Bristowe).

**Genito-urinary Tract.**—Renal congestion, and even acute nephritis, may appear as a complication. A case of cystitis with hematuria has also been reported (Comby and Le Gendre).

The **diagnosis** of influenza except in ill-defined, sporadic cases rarely presents serious difficulty. In obscure cases the discharges should be studied bacteriologically.

(a) **Climatic catarrhal affections** are sometimes hard to discriminate from sporadic cases of influenza. The former are usually attributed to sudden and great vicissitudes of temperature or exposure to strong drafts of air, while the latter come on independently of seasons of the year and of such agencies. Again, in influenza we usually observe the general features (nervous symptoms and debility) outweighing the local (catarrhal manifestations). Simple catarrhs do not tend to traverse the entire system with the same remarkable rapidity as influenzal catarrhs.

(b) **Typhoid fever**, particularly in its early stages, is often closely simulated by influenza with intestinal symptoms. Danger of confounding these two affections can be averted by remembering the facts that influenza gives a different history, begins suddenly, does not have the typical temperature-curve of typhoid, may present splenic enlargement—but by no means to the same extent as typhoid—and has no characteristic eruption.

(c) **Pneumonia** has quite frequently been mistaken for influenza, and especially when the thoracic symptoms in the latter have been unusually distinct. As already stated, lobar pneumonia may early complicate influenza in rare instances; but pneumonia is generally unilateral, while the lung-involvement in influenza is generally bilateral. In the former the physical signs indicative of consolidation are clearly marked; in the latter (unassociated with pneumonia) we often meet with those suggestive of congestive edema (impaired resonance, stationary crepitant and subcrepitant râles). The general features also present dissimilarities. Thus the nervous depression and the myalgic and neuralgic pains are more marked in influenza, while the pulse and respiration are apt to be less frequent than in pneumonia.

(d) **Cerebro-spinal meningitis** may manifest features that are almost identical with those characteristic of influenza. Thus during certain epidemics many “grippe” patients suffer from intense headache—occipital and frontal—rachialgia, fever, prostration, delirium, and stiffness of the muscles, with slight retraction of the head. There may be convulsions and vomiting at the outset. Here the history with reference to the character of the prevailing epidemic and the attendant circum-



stances must be carefully considered, but an absolute diagnosis is sometimes impossible unless a bacteriologic investigation of the discharges be made.

**Sequelæ.**—Among the pulmonary sequelæ are phthisis, chronic bronchitis, abscess and gangrene of the lungs (the latter two being rare), tachycardia, and angina pectoris. Chronic gastro-intestinal catarrh is not rare as a remnant of the acute form when the latter arises during the influenzal attack. Chronic nephritis, and less frequently cystitis, may also be mentioned.

Among *nervous* sequelæ which are both numerous and important are to be noted especially insomnia, neuralgia, migraine, melancholia, mania, meningitis, acute ascending myelitis, locomotor ataxia, peripheral neuritis, and perineuritis. The organs of special sense manifest a great variety of sequelæ, such as otitis media, otitis interna, mastoid abscess, conjunctivitis, keratitis, iritis, irido-chorioiditis, acute glaucoma, paralysis of accommodation, etc.

**Prognosis.**—The prognosis is, on the whole, good. Almost all fatalities are due to complications, especially *pneumonia*, and, less frequently, pulmonary congestion and edema, pleurisy, pericarditis, cerebro-spinal meningitis, etc.

The *circumstances connected with the individual case* often affect the outcome. Thus influenza runs a more severe course, and hence offers a correspondingly more serious prognosis in the very young, the very old, and those enfeebled on account of previous chronic disease (phthisis, valvular disease of the heart, emphysema, nephritis, etc.) than at other periods of life. During severe epidemics of influenza the mortality-list in the latter diseases is considerably augmented. Though epidemics vary as regards the mortality, the general average death-rate is a little under 1 per cent. In some epidemics it may reach 2 per cent., while in others it may be less than  $\frac{1}{2}$  of 1 per cent.

**Duration.**—The duration of the attack is brief, though subject to variations. In mild forms it is from two to four days, in the severe from seven to ten days; but complications and previous infirmities may greatly prolong the attack. The *duration of particular epidemics* rarely exceeds from four to six weeks.

**Treatment.**—**Prophylaxis.**—Experience has shown almost conclusively that the various drugs which have been counselled for their preventive effect (quinin, salicin, etc.) are devoid of value. The strongest persons are not immune, and those who are at either extreme of life or who are enfeebled by chronic organic disease should be most carefully protected by proper wearing apparel, and should not be exposed to the direct influence of the changes of weather. In this way we may hope to lessen, in a measure at least, the totality of the cases, since the inmates of hospitals and prisons have been known to escape absolutely when the community all around them was suffering from the disease. **Isolation** should therefore be carried out in hospitals and, under certain conditions, in private families, especially when the disease appears in households in which there are young children and aged persons. **Disinfection** of the catarrhal discharges, particularly the bronchial, which, as a rule, abound in the bacilli of Pfeiffer, must be rigidly carried forward.

**Treatment of the Attack.**—In considering the treatment of the attack the cases may be grouped under three heads:

(a) **Mild or Rudimentary Form.**—The cases belonging to this type require little besides careful hygienic management. However light the attack, the patient should remain in-doors and, if languid or prostrated, in bed for a period of two or three days. The *diet* should be light and nutritious (milk, eggs, rice, gruels, fresh vegetables, stewed fruit, etc.), and cooling drinks are to be preferred to hot ones, among the former lemonade or cold oatmeal water with lemon, and effervescent mineral waters (Apollinaris, lithia, Seltzer) being the best. The bowels should be moved regularly, avoiding, however, all purgation. Stimulants are not needful, though well borne as a rule, and the use of light wines is not objectionable if desired by the patient. In all cases of influenza, even of the mildest grade, I prescribe moderate doses of quinin (gr. iv—0.2592, three or four times daily), and if there be much headache this drug may be combined with Dover's powder and monobromate of camphor (of the first two gr. iij—0.194, each, and of the last gr. j—0.0648, in capsule), the dose to be repeated at intervals of three or four hours. To overcome the languor and debility, which are marked, I have found nothing so successful as strychnin.

(b) **Cases of Medium Severity.**—*General Management.*—This class of influenza patients betake themselves to bed, and should be kept there till convalescence is well advanced. During the febrile period the diet must be light, liquid, yet nutritious, and the food should be given every two or three hours. Although the patient has no desire for food, he should be urged to eat with regularity, no matter how small the quantity at each feeding. Moderate stimulation is also useful.

The *medicinal treatment* is, for the most part, simple and symptomatic. The neuralgia and myalgia may be relieved by the use of quinin, Dover's powder, and camphor, as before stated, but if the pain be intense, morphin administered subcutaneously is sometimes required. The temperature is somewhat reduced by these remedies, and especially by the quinin and Dover's powder, the latter of which acts as a diaphoretic. In addition, I am in the habit of ordering cool sponge-baths at intervals of two or three hours if the temperature be above 102° F. (38.8° C.). If not controlled in this manner, we may combine with quinin some antiseptic, such as salicylic acid or salol. I have found it necessary to add to the foregoing small doses of phenacetin (gr. ij—0.129), or acetanilid (gr. ij—0.129), the former being preferred, since it is superior to acetanilid in controlling insomnia, which is so often a troublesome symptom. *Sleeplessness* may, however, demand other and more potent hypnotics, such as sulfonal, chloralamid, opium, etc.

The local catarrhal conditions (coryza, laryngo-bronchial irritation, true bronchitis, etc.) must be treated according to the special indications presented in individual cases. For the coryza inunctions of animal fats over the forehead and bridge of the nose are useful. A flannel cap may be worn if agreeable to the patient. Steam inhalations through the nares and mouth often act beneficially, both upon the coryza and laryngo-bronchial irritation. For the latter common condition the following formula will be found serviceable:

R. Codeinæ sulph., gr. iv (0.259);  
 Ammon. chloridi, ʒv (20.0);  
 Syr. prun. virgin., fʒij (60.0);  
 Spts. junip. comp., q. s. ad fʒiv (120.0).—M.  
 Sig. One teaspoonful every two or three hours.

If this prescription fail to mitigate the cough, we may resort to morphin hypodermically, but always in small doses. In the later stages, particularly if bronchitis be associated with free secretions, the oil of eucalyptus (ʒiij to v—0.199 to 0.333), in capsule, every four hours, has in my experience proved useful. To obviate pulmonary complications I have been much gratified with the results from the use of strychnin (gr.  $\frac{1}{30}$ —0.0021), combined with vin Mariani (ʒss—16.0) at intervals of three or four hours. Chest-pains may be relieved by the use of turpentine stupes and sinapisms, both of which agents are also valuable in averting the more serious complications.

(c) **Severe Forms.**—*The general management* is similar to that recommended in cases of medium severity, excepting that freer stimulation is usually demanded. The medicinal treatment must also be more active than in the previous form, and often is heroic. Especially must quinin be given in full doses and continued, since it not only serves to reduce the temperature somewhat, but also to control the nervous symptoms and lessen the tendency to inflammatory complications. Nothing that exerts a depressing effect should be thought of, since the cardiac as well as the respiratory forces, must be conserved. Should there be sudden cardiac failure, it must be promptly met by the various forms of stimulants, including strychnin and the cardiac tonics. In addition to alcoholic stimulants, the aromatic spirits of ammonia is usually borne well, and should be administered. Strychnin must be given in full doses hypodermically every third or fourth hour. The various inflammatory complications that may arise must be treated as under other circumstances.

*The Convalescence.*—In all grades of cases the convalescence from influenza demands most rigid supervision, and the greatest injury to patients at this time comes from going out too early. Usually the temperature is subnormal for several days—a circumstance due to the weakness of the patient—and so long as this condition obtains the patient is highly susceptible to a chill. Hence it is a good rule not to allow exposure to the external changes of temperature until the temperature has been normal for several days. The diet should now be more liberal, and tonics, such as gentian, iron, and quinin, may be administered and continued until complete restoration of the patient's health has taken place. In every way possible exposure to reinfection during the period of convalescence is to be avoided.

The treatment of sequelæ must be conducted according to general rules.



## DENGUE.

*(Break-bone Fever.)*

**Definition.**—An acute infectious disease occurring epidemically in tropical and subtropical countries. Its chief symptoms are—a double febrile paroxysm (separated by an interval), arthritic and muscular pains, and a skin-eruption in about one-half the cases.

**Historic Note.**—The disease was prevalent in Java as early as 1779, in India in 1824, and later in the West Indies, Spain, and in some of the southern American States. Mild epidemics have visited Philadelphia, New York, and Boston, but, as a rule, it has not traversed regions beyond 32° N. latitude.

Its pathology has not been studied, death being the rarest of events.

**Etiology.**—McLaughlin of Texas has isolated from the blood and cultivated a micrococcus which he claims is the specific cause of the disease.

**Predisposing Factors.**—Its prevalence is favored by the summer season, and also to a slight extent by faulty hygienic conditions. On the other hand, age, race, sex, and social status are all without effect, most persons being susceptible; and, according to Matas, primary attacks are not protective in character, while other authors contend that they even predispose to subsequent ones. The epidemics spread along lines of travel by land and sea, and most authors agree that the disease is contagious.

**Clinical History.**—There is a period of incubation that lasts about four days and exhibits no prodromes.

**Invasion** then is abrupt with a slight chill; fever follows, the temperature reaching its maximum—103° to 106° F. (39.4° to 41° C.) or over—at the end of the first or on the second day, and is accompanied by headache and by muscular and arthritic pains. The patient's sufferings are intense, the pains being described as "breaking"—a peculiarity to which the disease owes the popular name of "*break-bone fever*." The joints become red, swollen, and very tender to the touch. The respirations and pulse are much quickened; there are anorexia and sometimes slight nausea. Febrile albuminuria is rare, delirium and mental torpor also; but prostration may become marked, and an erythematous eruption commonly appears. DeBrun<sup>1</sup> noted carefully the symptoms of dengue during the epidemic at Beirut (1892), and states that the eruption is roseolar, morbilliform, scarlatinous, or papular. He distinguished three groups of cases: 1. With high fever and marked associated symptoms, and with eruption; 2. Fever absent, the symptoms mild, with eruption; 3. The eruption the only symptom. The eruption may appear early, but has no fixed time, is evanescent in mild cases, and is never constant in character. It is attended with burning and itching, and DeBrun noticed desquamation of a varying intensity. Hemorrhages from the various organs (nose, gums, stomach, bowels, lungs, kidneys, etc.) may occur, and reach even a dangerous extent. The lymphatic glands are often swollen; the mucosæ of the nose and throat are hyperemic; the eyes are congested and the face flushed.

The initial fever lasts three or four days, and ends with a deep

<sup>1</sup> *Rev. de Méd.*, No. 6, 1894.

remission accompanied by profuse sweating. All the symptoms now vanish save a slight soreness and stiffness, but after an interval of two or three days the characteristic symptoms (including the eruption) reappear. This second febrile paroxysm is usually milder and shorter than the first.

The **duration** of the disease is from seven to ten days, the attack being followed by a slow convalescence, which may be interrupted by a relapse. The slowness of the recovery is due to persistent mental depression and marked physical prostration.

**Complications.**—Meningitis has been noted, but in extremely rare cases. Convulsions sometimes occur in children, and severe catarrhal inflammations of certain mucosæ (bronchial, gastric, etc.) may develop. Insomnia is common. Hyperpyrexia and pericarditis occur very exceptionally.

**Diagnosis.**—The diagnosis of the usual form of the disease (epidemic) is an easy one after observation of the first few cases, but a more difficult task is the discrimination of sporadic cases from *rheumatism*. The course and degree of the fever, however, differ in dengue and in rheumatism, while the eruption belongs to the former alone. *Influenza* in many of its manifestations resembles dengue very closely, and the differential diagnosis is a difficult one. In the former condition the herpes is usually the only eruption; the joints are rarely involved; there is neither a remission nor a recurrence of the fever; and serious complications are more frequent. The discovery of the bacillus of influenza is of course decisive, and the existence of an epidemic of either condition strongly suggests the true nature of the disease. *Scarlet fever* has an erythematous eruption, but the fever is continuous and the arthritic symptoms are usually wanting. *Yellow fever* has been mistaken for dengue, but is characterized by a single paroxysm; jaundice, black vomit, albuminuria, and grave nervous phenomena are features that are never seen in dengue.

The **prognosis** is with rare exceptions favorable, dangers arising only from the extremely rare serious complications.

**Treatment.**—The case presents a double indication: (*a*) to harbor the patient's strength, and (*b*) to meet certain leading symptoms. The first is to be accomplished by enjoining rest in bed, by a generous though carefully regulated diet, and by the timely use of stimulants and tonics. Among the symptoms that demand treatment is the fever, and when this is very high hydrotherapy is indicated. When moderate, cold sponging of the general surface, conjoined with internal antipyretics (phenacetin, acetanilid) in moderate doses, may be resorted to. For the intolerable pains morphin is to be administered hypodermically, this remedy often relieving the insomnia at the same time. If not, chloralamid and the bromids should be tried. Convalescence may be hastened by a suitable change of air.

## THE PLAGUE.

*(Bubonic Plague; Black Death.)*

**Definition.**—A specific contagious disease, occurring chiefly in unsanitary surroundings and characterized by high fever, cutaneous symptoms (petechiæ, etc.), and later by an inflammatory enlargement of the lymphatic glands (buboes). It occurs in epidemics.

**Historic Note.**—The plague is an Oriental disease, and has long been endemic in certain parts of India. Most European countries have in the past been visited by epidemics of the malady, but it is at present writing almost solely confined to its native habitat (India), to South China, and to parts of Asia. In May, 1894, a severe epidemic prevailed in Canton and Hong-Kong, to which cities it was imported from Northern India, and in the latter part of 1896 and the early months of 1897 another swept over India with devastating results.

**Etiology.**—Kitasato and Yersin both discovered during the epidemic at Hong-Kong a special bacillus which is probably the cause of the disease. It stains deeply at the ends, giving the appearance of a pair of micrococci, but is really a short rod-bacillus with rounded ends. Pure cultures are readily made, and when animals are inoculated with these the clinical characteristics of the disease are produced.

**Modes of Entrance into the Body.**—According to Kitasato, the bacillus enters either the digestive or the respiratory tract or by means of excoriations of the surface.

**Predisposing Causes.**—These are embraced in the single phrase—unhygienic conditions. It is safe to assert that without these fostering influences the plague would not prevail.

**Clinical History.**—The incubation period lasts from two to five or six days, and among prominent invasion symptoms are intense pains in the head, back, and limbs, and a dizziness causing the patient to walk with a staggering gait as if intoxicated. The temperature rises rapidly, sometimes to a hyperpyrexial level, preceded usually by slight shiverings or a chill, and delirium sets in early. There are torturing thirst, anorexia, and not rarely nausea and vomiting. Petechiæ, ecchymoses, and, in malignant types, hemorrhage from the stomach, bowels, and kidneys, may occur, and soon a typhoid condition develops with a marked tendency to circulatory collapse.

At the end of two or three days, if life be spared until then, buboes appear, and form the most marked and characteristic accompaniment of the disease. The inguinal and femoral glands are most generally swollen and inflamed; the axillary, submaxillary, etc. less frequently. This glandular enlargement may terminate in (a) resolution, (b) suppuration, (c) gangrene (rarely). Suppuration is generally favorable in import. Carbuncles may attend, but are comparatively rare.

**Diagnosis.**—The disease has been mistaken for *typhus fever*, but in the latter disease there is an absence of the early tendency to collapse, the characteristic buboes, carbuncles, extensive petechiæ, and hemorrhages. On the other hand, in the plague there is an absence of the characteristic typhus eruption. The geographic limitations of the plague should be borne in remembrance.



**Duration and Prognosis.**—The duration is brief—from three or four to eight or ten days—and extensive suppuration may prolong the attack. The prognosis is a very grave one, and the disease may prove fatal, like cholera, within a few hours.

**Treatment.**—This is largely preventive. All hygienic defects are to be corrected as quickly as possible, especially inadequate sewage, unclean surroundings, and impure water-supply. Isolation of the sick and thorough disinfection of the sick-room, the bed, and bed-linen, the vomitus, and the stools, are matters of paramount importance. Kitasato advocates steaming the bedding at 212° F. (100° C.) for one hour, or exposure for a few hours to sunlight, and that all infected articles be burned. “After recovery the patient is to be kept in isolation at least one month.”

The diet should be liquid, concentrated, and nourishing, and stimulants are demanded to obviate collapse. So far as known, medicines do not exercise any controlling influence, and hence they are used merely to combat the symptoms as they arise. As it is possible, however, to render animals immune to the disease, it is reasonable to anticipate that an antitoxic serum will soon be available for treatment.

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## ERYSIPELAS.

(*St. Anthony's Fire.*)

**Definition.**—A specific, acute contagious disease, characterized by a special inflammation of the skin and subcutaneous tissues, with a tendency to spread, high fever, moderate prostration, a disposition to mixed infection (suppuration, gangrene, etc.), and an average duration of fourteen days. It usually occurs in persons under forty years of age, and commonly in endemic, though also in epidemic, form.

**Pathology.**—Erysipelas is a specific inflammation involving the skin, subcutaneous and mucous surfaces, but the latter far less frequently. If uncomplicated, no other structures are involved. When inflammation extends to the subcutaneous connective tissue, there follows, as a rule, suppuration. The claim, however, that the inflammation may penetrate the skull and attack the meninges lacks convincing proof. Osler in one case traced the extension from the face along the fifth cranial nerve to the meninges, where an acute meningitis and thrombosis of the lateral sinuses were excited. The specific cocci are found in the superficial lymph-vessels and spaces of the affected skin, being most abundant in the ever-advancing elevated margin. Beyond the border of the inflamed region they occupy chiefly the lymph-vessels, where they are finally overpowered by the phagocytic action of the leukocytes. Microscopic examination of the involved area reveals the changes of simple inflammation.

**Etiology.**—**Bacteriology.**—The specific cause of the disease is the *streptococcus erysipelatis* of Fehleisen, which is probably identical with the ordinary pus-producing streptococcus. Fränkel and Kirchner have

investigated, experimentally, the streptococcus of erysipelas, and contend that their results offer convincing proof of the separate identity of the streptococcus pyogenes; but most observers hold that the latter cannot be distinguished from the *streptococcus erysipelatis* by any known test. The streptococci of erysipelas assume the form of a serpent or chain (chain-forming coccus of Cohn), and are very small, somewhat variable in size, and thrive on all kinds of culture-media. Their favorite situations are the lymph-vessels and the cutaneous connective tissue, where they are found in colonies composed of myriads of cocci. They are rarely found in the blood-vessels, and in blood-serum they are caused to disappear by the action of the phagocytes; yet in exceptional cases intra-uterine infection has occurred.

That the streptococcus erysipelatis is a saprophytic organism is shown by the fact that the identical cocci have been discovered in inanimate and decomposing animal and vegetable substances.

**Predisposing Causes.**—Among the most important disposing causes of erysipelas are—

(1) **Season.**—In a paper on “Seasonal Influences in Erysipelas, with Statistics”<sup>1</sup> I have shown, as the result of an analysis of 2010 cases collected from different sources, that the various seasons of the year exercise a potent influence upon the frequency of this affection. Thus month by month the cases increase, in slightly varying ratio, from August to April, the latter month giving the greatest number, and then there is a rapid decrease from April to August, when we find the smallest number. Again, one-half of all the cases occur during the months of February, March, April, and May, and 15.9 per cent. during the month of April alone. It would appear that the winter and spring months, though more particularly the latter, increase the susceptibility to this disease. It was found that a low barometer and mean relative humidity invariably correspond with the annual period in which the greatest number of cases occur, and that the highest percentage of relative humidity corresponds with the months affording the fewest cases.

(2) **Age.**—From the notes of 1894 cases I found that in 25.8 per cent. the age of the patient was between twenty and thirty years. From thirty to fifty years the cases slowly decreased in number, and after fifty years quite rapidly, while more than 15 per cent. of the cases occurred before the age of twenty. The great liability of newly-born infants is well known.

(3) **Sex.**—This factor was noted in 1767 cases, and a marked preponderance of the male over the female sex was noted (about 3 to 2).

(4) **Previous Attacks.**—Of 450 cases, there had been previous attacks in 39 (8.6 per cent.), in one instance, four, and in another seven, antecedent attacks having occurred, while second and third recurrences were not uncommon.

(5) **Family predisposition** exercises a slight though decided influence. It was noted in 4 of the 450 cases.

(6) **Certain Antecedent Affections.**—Dr. M. Booth Miller examined the history of 301 cases, and found that acute coryza preceded the attack in 13 instances. Slight lesions of the Schneiderian mucous membrane may be assumed to exist in such instances, offering a condi-

<sup>1</sup> *Proceedings of the American Climatological Association*, 1893.



tion favorable to specific infection (*vide infra*). Testimony confirming the now well-known fact that certain chronic diseases (chronic Bright's, phthisis, organic heart disease, chronic alcoholism, syphilis, etc.) augment a receptivity to the complaint has also been brought to light by my own researches.

(7) **Slight Injuries, Abrasions, etc.**—Erysipelas will not develop on a surface which does not present a break, but with this present may do so though the latter be so trivial as to escape observation. Slight abrasions and fissures, either in the mucous membrane of the nose or in the skin of the face or ear, as well as all forms of slight injuries, are liable to furnish a path of ingress to the specific organism. Yet in 643 out of the 2010 cases mentioned above and examined with reference to this point, previous lesions were noted in but 13. Women who have been recently delivered and persons subjected to surgical operations are peculiarly liable, and any deeply-seated focus of irritation, as an area of necrotic bone or a chronic abscess, such as sometimes occurs in suppurative arthritis, appendicitis, etc., may give rise to repeated outbursts of erysipelas.

(8) **Antihygienic Surroundings.**—These doubtless predispose to the affection, as has been shown by the prevalence of erysipelas in hospitals and institutions in which the sanitary arrangements are markedly faulty.

**Modes of Conveyance of the Contagion.**—The latter may be air-borne. It has been collected from the air of rooms and wards occupied by erysipelas patients; but to what distance and precisely under what circumstances it can be conveyed is not definitely known. It may also be transferred for a longer or shorter distance by fomites, by instruments, unclean hands, etc., the *infection* being a result either of *contagion* or *inoculation*, and the direct avenue of entrance for the specific coccus being a break in the skin-surface or mucous membranes.

**Clinical History.**—I shall discuss only the so-called medical or idiopathic erysipelas, the traumatic variety falling more properly within the domain of surgical treatises.

**Incubation.**—This is somewhat varied, though it ranges usually from seven to fourteen days, and the prodromal symptoms are, for the most part, general in character, consisting in headache, restlessness, cough and sore throat, anorexia, and general slight or moderate pyrexia. These endure for a very variable period—from a few hours to several days—when the invasion with its characteristic features develops.

**Invasion Stage.**—The symptoms are—(1) local and (2) general.

(1) At first the affected part feels hot, tense, painful, and is tender to the touch. Very soon a small circumscribed area becomes red, swollen, firm, and shining, and simultaneously the subjective symptoms (pain, heat, etc.) become aggravated. The point of election is usually on the nose, but it may be on the ear, the face, or elsewhere about the head, and thence the inflamed, swollen zone spreads, chiefly in the direction of one or the other side of the head. Separating the diseased from the unaffected skin there is a sharp line of demarcation in the form of an elevated brawny ridge, which can be seen and felt. While the inflammation is advancing there may be noted, beyond the border of the latter, little red streaks and spots that grow in area till at last they become confluent. The degree of redness increases in intensity as the case ad-



vances, but any natural prominence or fold in the integument may prevent further extension of the inflammation (*e. g.* naso-labial folds, border of the hairy scalp, etc.). In cases of average severity the face is much swollen, the eyes closed on account of tumefaction of the eyelids, the ears greatly enlarged (far better marked on one side than the other, as a rule), the scalp swollen and tender, and the facial lineaments often changed beyond recognition. In a minority of the cases the inflammatory process extends from the head to the arms, to the trunk, and even to the lower extremities (*erysipelas migrans*), and in such instances the face may be healed while the disease is yet extending over other portions of the body. Even in the ordinary form, which is usually confined to the face, ears, and portions of the scalp, those parts first affected pale while yet the local inflammation is extending its boundary lines. When the progress of the inflammatory process has become arrested the peripheral ridge ceases to extend and grows pale; the inflammation then subsides, and finally disappears altogether.

The epidermal layer may become elevated over circumscribed areas, giving rise to larger or smaller vesicles or bullæ (*erysipelas vesiculosum*). Suppuration may attack these large vesicles, whereupon they fill with pus (*erysipelas pustulosum*). As the result of intense infiltration the part or parts may become gangrenous—*erysipelas gangrænosum*. Enlargement of the cerebral lymph-glands is also common. Desquamation follows erysipelas, and the face often presents a more delicate complexion than before the occurrence of the attack.

(2) **General Symptoms.**—With the onset of the local disturbances or even somewhat earlier, the patient is seized with repeated fits of chilliness or shivering, or, less commonly, there may be a severe rigor. Immediately, and more rapidly than before, the temperature rises to a height of 104° or 105° F. (40°–40.5° C.) on the evening of the first day. As a rule, the temperature reaches its maximum (105° to 107° F.—40.5° to 41.6° C.) on the third evening. Marked nocturnal remissions of temperature (2° to 5° F.—1.1° to 2.7° C.) are the rule, but the evening temperature may in rare instances be to an equal degree lower than the morning. In a week from the appearance of the eruption the temperature declines rapidly to normal, and usually within twenty-four or thirty-six hours; sometimes, however, the course of the fever is much more prolonged and defervescence may be less critical. In *erysipelas migrans* a long and decidedly irregular temperature-curve is presented, the period of decline also showing many deviations from the normal curve when complications are present. The pulse is frequent, of good volume, and soft. I have been able to confirm the observations of DaCosta, Strümpell, and others that the cutaneous inflammation in erysipelas (particularly *erysipelas migrans*) may advance to a slight extent even after the temperature has returned to the normal grade.

The tongue is furred, the anorexia intensified, and there are apt to be nausea and frequent vomiting. The bowels are usually constipated, though I have observed a few instances in which marked diarrhœa developed late in the attack. The inflammation may extend to the mucous membrane of the throat and larynx, causing swelling and edema of the parts. It may also involve the serous membranes, though rarely. The nervous symptoms may or may not be conspicuous, but there are apt to

be intense headache and restlessness, with some mental aberration at night. Actual nocturnal delirium appears in the severer forms, and in erysipelas occurring in drunkards delirium tremens may suddenly develop. The *urine* presents the usual febrile characters (high color with increased urea and diminished chlorids). Quite commonly it contains a little albumin, and rarely acute nephritis occurs as a complication.

Abundant observation has shown that there is a direct correspondence between the intensity of the local and constitutional disturbances in this disease.

Often in severe types (such as are apt to arise in old, much enfeebled, or intemperate persons) of facial erysipelas the typhoid (adynamic) condition is developed. The tongue is dry and brown, the lips and teeth are covered with sordes, the pulse grows very rapid and feeble, and the bowels are apt to be loose. Ataxic nervous symptoms show themselves.

**Complications and Varieties.**—An analysis of 1674 cases of erysipelas with particular reference to complications gave an interesting series of results, and one at variance with the notions of most authors. Some are given here briefly, the complicating conditions being placed in the order of frequency of occurrence: Abscess, 105; rheumatism, 20; delirium tremens, 10; lobar pneumonia, active delirium, phlebitis, pleurisy, each 7; acute nephritis, 6; synovitis and diarrhea, each 5; tonsillitis, 3; catarrhal pneumonia, otitis media, pharyngitis, edema of the larynx, acute bronchitis, each 2; endocarditis, meningitis, each 1.<sup>1</sup>

The fact that acute articular rheumatism is a relatively frequent complication of erysipelas is worthy of special notice, for the reason that the attention of the profession has not hitherto been called to it. The symptoms of rheumatism usually come on several days after the onset of erysipelas. "So long as the specific agent upon which rheumatism depends is not known, so long must we remain in ignorance of the true explanation of this combination of diseases." In a few instances pneumonia appeared early, being due most probably to special localizations of the specific streptococcus. To such cases the term "pneumo-erysipelas" may be appropriately applied. The cases—2 in number—in which acute nephritis developed during the first few days of the attack should in like manner be termed "nephro-erysipelas." With few exceptions, however, the complications enumerated are secondary affections. Meningitis was present in a single instance only, and hence active delirium in this disease points to a severe type of infection, but not to meningitis as a rule. Many different varieties have been described.

Apart from those already referred to more or less at length (cutaneous, gangrenous, vesicular, "pneumo-," "nephro-," and migratory erysipelas), two other forms—namely, *phlegmonous*, or *cellulo-cutaneous*, and *relapsing* erysipelas—should be mentioned. The former exhibits an inflammation of the subcutaneous connective tissue which tends to suppurate, and a glance at the complications of the affection shows that suppuration occurs with the greatest frequency among complicating conditions. But the so-called phlegmonous erysipelas cannot be properly

<sup>1</sup> "The Complicating Conditions, Associated Diseases, and Mortality-rate in Erysipelas," by the Author: *The Int. Med. Mag.* for Oct., 1893.



regarded as a distinct type, since the atypical manifestations are due to secondary infection with the pyogenic organism. Relapsing erysipelas, however, constitutes the chronic form of the disease, recurring at longer or shorter intervals, and usually in the same locality. It is commonly due to some deep-seated focus of suppuration.

**Sequelæ.**—The hair often falls, but it is usually replaced by a fresh crop. Abscesses in various parts of the body, particularly the eyelids, are of common occurrence (*vide* Complications), and chronic otitis media and chronic nephritis may date from an attack of erysipelas. *Per contra*, erysipelas is reputed to be curative of certain affections (eczema, lupus, carcinoma, sarcoma, rheumatism, etc.).

Out of 476 cases collected by me relapses occurred in 54 (11.3 per cent.), and in 1 of these instances 5 relapses occurred; in 2 others, 4; and in 3 patients, 3. First and second relapses were still more common.<sup>1</sup>

The **diagnosis** is made with ease after the eruption has fully developed, and its appearance, seat, and behavior, particularly the manner of extension of the brawny, ridge-like edge (best marked on the forehead), are the features that should serve to distinguish it positively from every other disease.

First, *erythema* produces superficial redness, but is not attended with heat, swelling, or fever. *Urticaria* assumes the form of pale-red circular wheals, which cause marked itching and appear in successive crops, often disappearing in the course of a few hours. *Acute eczema* of the face, when intense, may somewhat resemble erysipelas, but it lacks the peculiar border and mode of progression so characteristic of the latter disease. Again, eczema produces particularly troublesome itching, and the swelling is less than in erysipelas. *Chronic erythematous eczema* is met with later in life, is without fever, without any considerable swelling or pain, while, on the other hand, it excites intense itching. *Eczema nodosum* is characterized by its nodosities at their usual seat in the vicinity of joints.

**Course and Duration.**—In my own experience, based upon 1880 cases,<sup>2</sup> the average duration (including the prodromal stage and period of convalescence) in persons under forty years of age is fourteen days. The course of the disease is much lengthened by complications or by the pre-existence of chronic affections, and increases with age after the fiftieth year.

The **prognosis** is favorable, and it is rare for erysipelas to assume a malignant type. Perhaps the chief dangers lie in certain complications, especially extensive suppuration, pneumonia, acute nephritis, delirium tremens, etc. Acute articular rheumatism, though relatively frequent, is comparatively harmless; but *previous debility*, especially if dependent upon chronic diseases, as syphilis, chronic rheumatism, gout, tuberculosis, organic disease of the heart, and the like, increases the percentage of deaths considerably. Again, age has a positive influence upon the mortality, which it augments moderately after the forty-fifth year, and most decidedly after the sixtieth year. When death occurs it is due to exhaustion.

<sup>1</sup> *Journal of the American Medical Association*, July 22, 1893.

<sup>2</sup> "Points in the Etiology and Clinical History of Erysipelas," by the Author: *Journal of the Am. Med. Assoc.*, July 22, 1893.



The *mortality-rate* is low, as shown by the results of my own collective investigations into the subject.<sup>1</sup> I found the general average death-rate to be 5.6 per cent., while in cases from private practice it was 4 per cent. In persons under forty years of age it was only 3.5 per cent., while in those over seventy years it was 46 per cent. The traumatic cases gave a mortality of 14.5 per cent.

**Treatment.**—The treatment of erysipelas falls naturally into three subdivisions: (1) Dietetic; (2) Constitutional; (3) Local.

(1) **Dietetic.**—Proper attention to the diet is of the first importance. It must be generous and composed of highly nutritious articles, and if the temperature be high, only liquid forms of nourishment should be administered in definite quantities and at regular, brief intervals. Rectal alimentation should be resorted to if the stomach rejects a suitable dietary, and I feel confident of the fact that liberal feeding is of greater service to the patient than any of the recognized forms of medicinal treatment. Lack of attention to the patient's diet during the primary attack tends to increase the frequency of relapse. In persons over fifty years of age, and in those in whom the vital processes have been lowered on account of previous chronic diseases, correct alimentation is of paramount importance, often abridging the otherwise much protracted course of the affection. There can be no question but that the typhoid state of the system met with in many cases of erysipelas is attributable, indirectly at least, to malnutrition. When nourishment is exhibited in a proper manner stimulants are rarely required.

(2) **Constitutional Treatment.**—When, despite an appropriate diet, the pulse becomes very rapid and feeble, the heart's first sound indistinct, and the tongue dry or brown, indications for the use of stimulants are present and must be heeded. Alcoholic stimulation is most apt to be required in the aged and in patients previously enfeebled by chronic disease. When needful, the alcoholics may be given with a comparatively free hand, 12 to 16 ounces (360.0–480.0) of whiskey daily in divided portions. Strychnin gives prompt results, and may be used in association with the alcoholics. In marked gastric irritability champagne is to be preferred.

The tincture of the chlorid of iron was first extensively used in this disease by English authorities, and was formerly regarded by most clinicians as a truly specific remedy. In 74 cases of erysipelas which were treated by this remedy alone, the average quantity being 1 dram (4.0) daily in divided doses, in the Pennsylvania Hospital by Drs. Lewis, DaCosta, Longstreth, Meigs, and others, the death-rate was 4 per cent.<sup>2</sup> At the present day the profession are, for the most part, agreed that other preparations of iron are at least equally efficacious. Quinin is a valuable remedy in erysipelas, and during the past twelve years I have employed it in not less than 30 cases, confining its use to instances in which the temperature touched 103° F. (39.4° C.), and, with a single exception, in uncomplicated cases (22 in number) the nocturnal remissions were decidedly greater and the evening exacerbations less marked. In every instance iron in some form was administered simultaneously. J. M. DaCosta first used pilocarpin in erysipelas at the Pennsylvania

<sup>1</sup> *Loc. cit.*, p. 3.

<sup>2</sup> "The Treatment of Erysipelas," by the Author: *Therapeutic Gazette*, July 16, 1894.

Hospital. His experience showed that when given hypodermically (gr.  $\frac{1}{6}$ —0.010) in the very early stage, and repeated three or four times at intervals of two or three hours, it often aborted the attack. If we except this use of the drug, it is only in cases attended with high temperature with slight morning falls that pilocarpin should be employed; and the condition of the pulse and heart can be relied upon as a guide to its administration. Whether or not the favorable results from the use of pilocarpin are to be ascribed to a property possessed by it of stimulating phagocytic action is not yet clear.

Numerous *antiseptic* remedies have been recommended, and I have for a decade and over been exhibiting mercuric chlorid in moderate-sized doses throughout the febrile stage, with some amelioration of the symptoms.

Of late various bacteriologists have been endeavoring to obtain a serum antagonistic to the toxin of erysipelas. Experimentally, the success has been all that could be desired, but it has been difficult to obtain a serum of sufficient potency for therapeutic purposes. The most successful is that of Marmorek, now manufactured extensively, and, although Petruschky has declared it useless, the results of earlier and later investigators (Bornemann and Mérieux) cannot be ignored, and it must be recognized as possessing considerable value.

Certain symptoms demand internal medication. When the fever, as sometimes happens, is alarmingly high, its reduction must be accomplished, and the best method is by means of cold spongings combined with the ice-cap, or cold or gradually cooled baths. The happy effects of this agent—cold—are manifold. Guaiacol has recently been employed for the purpose of reducing the temperature, and found highly efficacious. The tendency to spontaneous remission of fever in this disease must, however, be steadily borne in mind.

For *marked nervous phenomena*, such as pain, sleeplessness, and active delirium, hyoscin hydrobromate (gr.  $\frac{1}{100}$ —0.0006) has been tried hypodermically in numerous cases at the Medico-Chirurgical, Pennsylvania, and Philadelphia hospitals, and has given promise of being a valuable remedy. It should not be employed when the heart-power is found to be deficient, and to fulfil the same indications we may utilize the following: Sodium bromid, gr. v (0.324) every two hours, or gr. xx-xxx (1.296–1.944) at night; morphin, gr.  $\frac{1}{8}$  (0.008), and chloral, gr. x (0.648), in combination every half hour for three doses; potassium bromid, gr. x (0.648), and tincture of cannabis indica, ℥x (0.666), in combination at bed-time; atropin, gr.  $\frac{1}{80}$  (0.0008), and morphin, gr.  $\frac{1}{6}$  (0.0108), hypodermically.

The treatment of the various *complications* must be conducted in accordance with general principles applicable to each.

(3) **Local measures** have always held a prominent place in the treatment of erysipelas. The list of agents that have been used topically is long and embraces all classes of therapeutic substances. In the paper previously cited it is stated that in the three series of cases (247) that were treated at the Pennsylvania Hospital, together with a few collected from other sources, no less than fifty different remedies and preparations had been employed locally. Among those most frequently used were elm (37 cases); lead-water and laudanum (20 cases); carbolic acid (1 to



40), injected subcutaneously (18 cases); zinc oxid (14 cases); mercuric-chlorid solution (14 cases); ichthyol ointment with lanolin (8 cases), etc. Many of these preparations were prescribed for their effect in excluding the air—a leading indication. This I am in the habit of meeting by the use of carbolized vaselin or cool carbolized oil.

A knowledge of the microbic nature of erysipelas has led to the local application of numerous antiseptic remedies, and it is along this line that the greatest advances in the treatment of the disease are to be expected. Mention has been made of the method of injecting carbolic acid. Here the aim is to check the spread of the inflammatory process by inserting the needle at numerous points just beyond the inflamed border. The method (introduced by Heuter) has been much practised by Henry at the Philadelphia Hospital, and more recently by Osler at the Johns Hopkins Hospital, and is especially applicable in erysipelas migrans. In the statistics before given a solution of mercuric chlorid (1 : 4000) was used locally in 14 instances, to which I can add the results of 12 others at the Medico-Chirurgical Hospital and in private practice. In nearly all of the cases it was employed in the form of a lotion over the inflamed surface. In a few it was injected beneath the skin, as in the case of the carbolic acid. More recently it has been recommended to scarify the affected part and follow with the application of a solution of mercuric chlorid. In view of the fact that the streptococcus is found chiefly in the more superficial channels of the corium, it follows that it may be attacked directly by the mercuric-chlorid solution when the latter is used after scarification; and this method of treatment is at once most promising and rational. In 8 instances (3 of which have been previously reported) it was attended with brilliant results, limiting the spread and allaying the severity of the local inflammation. At the Pennsylvania Hospital uniformly good results were obtained from the local use of ichthyol ointment with lanolin (DaCosta and others). Zelewsky found ichthyol efficacious in every form of erysipelas, being superior to other remedies. He prescribed the agent as follows:

Ry. Ammon. sulpho-ichthyol,	
Spts. æther.,	āā. 1 part;
Collodii elastici,	2 parts.

Thomas advocates thorough rubbing of a strong ointment of ichthyol with vaselin or lanolin into the red area and into the adjoining healthy skin, covering the parts with lint or the ordinary surgical dressing. Whalen<sup>1</sup> has recently reported most strikingly favorable results in 4 cases of facial erysipelas from the use of external applications of guaiacol.

Many special modes of treatment in erysipelas have been brought forward recently, but of these only two are deemed worthy of brief notice: (a) Method of Koch. By means of a soft brush we apply a thin and regular covering of the following pomade:

Ry. Creolin,	1.0;
Iodoform,	4.0;
Lanolin,	10.0.

<sup>1</sup> *Journal of the American Medical Association*, April 28, 1894.



The parts are then covered with leaves of gutta-percha. (b) Method of Hallopeau.<sup>1</sup> A mask of several thicknesses of linen is soaked in a solution of sodium salicylate (1:20) and applied over the parts, which are then covered with rubber bands to prevent evaporation. Relief is said to be almost immediate, and a cure is had in from three to five days.

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## DIPHTHERIA.

(*Diphtheritis; Angina Maligna; Croup.*)

**Definition.**—An acute, contagious disease caused by the Klebs-Löffler bacillus, and characterized, anatomically, by a croupous-diphtheritic inflammation of the mucous membrane of the pharynx and upper air-passages. Clinically, it is characterized by irregular fever, prostration, and, frequently, albuminuria; also by the secondary development of toxemia, and often of croupous laryngitis, or cardiac failure. It is often followed by peculiar paralyses. In large municipalities it behaves endemically, and from time to time epidemically.

*Pseudo-diphtheria.*—There are forms of inflammation occurring most frequently in the pharynx and adjacent air-passages (and also in many other parts of the body) that are attended with the formation of a pseudo-membrane, and are not caused by the Klebs-Löffler bacillus. These cases have been studied exhaustively by Prudden and others, who have usually found the streptococcus to be the specific cause of infection. The latter, however, has been found occasionally in the pharynx of healthy children and in the inflamed mucous surfaces met with in erysipelas and measles. “Pseudo-diphtheria,” so called, is very common in scarlatina.

**Pathology.**—The true diphtheritic inflammation has for its chief pathologic peculiarity the production of a fibrinous exudate. When the inflammation is superficial and of a mild grade, a croupous membrane is produced which can be easily removed from the mucosa, which it covers. Its formation is accompanied by a necrotic process that does not extend below, but practically replaces the epithelial layer of the mucous membrane. In the severer types of the affection, however, the fibrinous membrane infiltrates all the layers of the mucosa, which undergoes necrosis more or less nearly complete. In the severest forms the submucous layer may also become necrotic. It is to be borne in mind that the production of the fibrinous exudate in croup or diphtheria is always preceded by coagulation-necrosis of the epithelium. The mucous membrane surrounding the exudate is hyperemic, more or less edematous, and the seat of muco-purulent secretions.

**The Pseudo-membrane.**—Its composition comprises fibrin, pus, disintegrated leukocytes, flakes of necrosed epithelium, bacilli, and sometimes red blood-corpuscles. The fibrin has two main sources: (a) “The fibrinogen of the inflammatory matter,” which transudes through the capillary walls; and (b) Disintegrated, migratory leukocytes, which form branching fibrillæ. Weigert holds that the inflammatory exuda-

<sup>1</sup> *Journal of the American Medical Association*, vol. xv. p. 334.

tion is coagulated by a ferment derived from the disintegrated leukocytes.

The Klebs-Löffler bacilli are found, chiefly and in varying relative numbers, in the meshes of the fibrillæ, but also in the granular fibrin and on the adjacent mucous membrane. Frequently other micro-organisms are associated (streptococci, staphylococci, etc.). The membrane presents a grayish-white color, and, if croupous in character, can, as before mentioned, easily be removed. When the mucosa is deeply involved the membrane is thicker, firmer, and more adherent, so that its removal entire cannot be effected without great difficulty, and without, as a rule, injury to the surface, as shown by bleeding, etc. The character of the pseudo-membrane is affected by the nature of the underlying structure; thus in the pharynx it is firmer and less easily separable than in the larynx and trachea, where a distinct basement membrane is found (Flexner). As the membrane becomes older its color is apt to grow darker, becoming yellow or even dark brown. It sometimes becomes gangrenous, and softens or disintegrates with the production of a very offensive brownish, semi-liquid excretion. The advancing edge of the false membrane is usually thin. On the other hand, when the process has become arrested the edge is apt to look raised or wrinkled, and later it may be distinctly curled up.

The membrane may extend downward into the ramifications of the bronchi. In such cases there is apt to be a lobular pneumonia, and this latter condition may occur without extension of the membrane. Occasionally there is a lobar pneumonia. A generalized bronchitis extending to the smaller bronchi is common from the irritation of aspirated substances. In rare cases the membrane has spread into the esophagus and even into the stomach.

After separation of a croupous membrane repair consists merely in a restoration of the epithelial layer—a process which is initiated by the fragments of epithelium that remain along the edges of the diseased area, and proceeds centrally. On the other hand, in true diphtheria, with necrosis more or less nearly complete of the mucosa and even the submucosa, sloughing occurs, and the missing structures are replaced by cicatricial tissues.

**The Heart.**—The muscular structure and the nervous mechanism suffer most. The histologic changes may be of the parenchymatous variety, but only in mild instances; whereas in severer cases fatty degeneration is conspicuous. In still other cases the chief pathologic characteristic is an interstitial myocarditis, and rarely the lesions of pericarditis and endocarditis have been noted. The heart is by no means always involved.

The *spleen* is commonly enlarged, though not to an excessive degree. The *blood* is dark, its coagulability is greatly diminished, and Canon and Frosch have in a few cases found the bacilli in the blood of those dying with diphtheria. The red-corpuscles are somewhat decreased in number during the course of the disease, while the white corpuscles are increased. Bouchut and Dulinsay consider the grade of leukocytosis of prognostic value, and claim that it varies directly with the severity. Grawitz has determined in numerous cases a higher specific gravity of the blood during diphtheria. The *lymphatic glands*

of the neck become swollen as a rule, but they show little tendency to suppurate.

**The Kidneys.**—The kidneys show degenerative changes, the usual *kidney-lesion* being a hyperemic swelling with edema of the interstitial tissues, and often hemorrhagic spots in the cortex. Sometimes there is a marked glomerulo-nephritis, and more rarely a diffuse granular degeneration of the epithelium. Minute areas of necrosis have been observed in the internal organs, in which fibrin has been found deposited (Oertel). Welch and Flexner have produced, by artificial inoculation upon guinea-pigs, kittens, and rabbits, foci of cell-death in the lymph-glands throughout the body, in the spleen, liver, lungs, heart, and intestinal mucosa. When the dose is small and the animal lives several weeks, the paralysis which belongs to the disease may develop.

The *nerves*, in cases of paralysis, have shown parenchymatous and interstitial inflammatory lesions. In paralysis of throat-muscles (*i. e.* those near the locality of the pseudo-membranous inflammation) the latter show also round-cell infiltration and fatty degeneration of the fibers.

The *glands*, especially of the neck, are often much enlarged, and there is, in pronouncedly septic cases in which a mixed infection is found by culture, a good deal of tumefaction of the neck, this sometimes even obliterating the normal contour from jaw to clavicle.

**Etiology.**—True diphtheria is caused by the Klebs-Löffler bacillus, and all cases of supposed diphtheria in which the bacillus is absent are to be regarded as non-diphtheritic. The etiologic is, therefore, quite different from the pathologic significance of this term. Recent researches have removed all doubt as to the specific nature of the Klebs-Löffler bacillus.

**Bacteriology.**—The bacillus diphtheriæ nearly equals in length that of the bacillus tuberculosis, and is twice the diameter of the latter. It has rounded extremities, which are also frequently bulbous, giving it the appearance of a dumb-bell. At times one end only is clubbed, or, more rarely, one or both ends appear pointed. The bacilli are immobile, do not form spores, and stain readily, the best agent being alkaline methyl-blue. Their manner of taking the stain is important. The bacilli show alternating segments of darker and lighter stained areas, and often minute dots showing a most intense and deep staining. They grow on most culture-media, but for clinical purposes Löffler's blood-serum is important (3 parts blood-serum and 1 part neutral or slightly alkaline nutritive bouillon, containing 1 per cent. of glucose). Inoculated on this, they outgrow all other organisms that may be present, and within eight hours or less show numerous spots, one-half to one millimeter in diameter, which have a dull surface and a dense white or somewhat yellowish color. There are usually present also smaller points which have different appearances and which are colonies of other organisms. The former are the colonies of the bacillus diphtheriæ, and from these microscopic preparations and (by further cultivation) fine cultures can be obtained. The bacilli are semi-anaërobic, and thrive at the temperature of the human body; a temperature of 122°–136.5° F. (50°–58° C.) causes their destruction in ten minutes.

*Pseudo-diphtheria Bacillus or Bacillus Xerosis.*—From many cases,



often showing no lesions, an organism may be obtained that is identical in appearance, manner of culture, growth, etc. with the bacillus diphtheriæ, but inoculation with it causes no lesions. The works of Abbott, Roux, Yersin, and others seem to show that this is an attenuated form of the true bacillus, and varying grades of pathogenicity may be found between the two. The distinction from the pathogenic bacillus can only be made by determining the lack of infection after inoculation.

**Site of Infection.**—In the human family the seat of election of the bacillus diphtheriæ is usually the faucial mucosa, and less frequently other abraded skin and mucous surfaces. The bacilli do not penetrate the mucosa, and hence do not find their way into the lymphatic or circulatory system, but remain at or very near the site of the local changes.

**The Toxins.**—Toxins are absorbed from the diseased spots by the lymphatics and blood-vessels, and produce the general phenomena in uncomplicated cases. They have been isolated from artificial cultivations of the microbe, and when inoculated the chief ptomain of the Klebs-Löffler bacillus so modifies the solids and liquids of the body as to render the subject immune (Behring). Another, however, if employed in like manner, produces dangerous and even fatal symptoms (convulsions, paralysis, etc.).

It is certain that the bacillus can maintain an existence for months outside of the body, though its usual habitat is unknown unless it be the organic constituents of the superficial soil. The virulence of its products is modified by many individual conditions, and chief among these is a healthy and intact condition of the mucous membranes, which greatly reduces the susceptibility to the disease.

**Associated Microbes.**—With the Klebs-Löffler bacillus are frequently found other microbes, especially streptococci and staphylococci. These pass beyond the site of local infection, reaching the internal viscera and other structures, and, as will be seen hereafter, give rise to the serious septic element of the disease.

**Modes of Infection.**—When the bacillus leaves the body of the sick it is contained in particles or shreds of the diphtheritic membrane, or in the expired air. Infection may then occur (a) *By direct contact* with the shreds of membrane thrown off—*e. g.* when the latter are ejected by coughing and lodge upon the conjunctivæ or faucial mucosa of bystanders. Under this category come the cases in which the deadly poison is transferred to the physician and attendants, with resulting infection, from the sucking of tracheotomy-tubes. (b) *By inhaling the air* surrounding the patient (contagion). Infection by contagion, however, does not extend beyond a radius of a few feet from the patient. (c) A very leading manner of conveyance of the bacillus from the sick to the healthy is by *fomites*. The contagion adheres tenaciously to a great variety of objects (toys, clothing, library books, letters, slates and drinking-cups in the public schools, etc.), and in this way the germs of diphtheria have been transferred over great distances and have given rise to the disease long after. The latter fact renders it difficult to trace certain cases to previous ones, to which they invariably owe their origin. (d) *Sewer gas, per se*, is to be regarded as non-pathogenic, or at least so far as this affection is concerned (Laws). It may, however, become a carrier of diphtheritic poison. (e) I regard it as highly

probable that the disease may be communicated by *domestic animals* (fowls, cats, etc.).

As to the exact conditions under which infection occurs, our knowledge is as yet incomplete. We know definitely the usual point of local infection in man, and also that a catarrhal mucosa or an open lesion of a mucous surface invites infection. It is not certain, however, that even a slight lesion of the mucous surface is essential to infection, though it is very questionable whether the diphtheritic germs ever find lodgement in the perfectly healthy mucosa. Some writers claim still that the Klebs-Löffler bacillus may enter the blood through the respiratory system and give rise to primary constitutional symptoms, the local manifestations in the throat being secondary. I have met with a single instance that would lend support to this view.

**Predisposing Factors.**—(1) *Age*.—This is the most important factor, diphtheria being, in the main, a disease of childhood. Most cases occur between the second and seventh years, while the receptivity diminishes rapidly after the tenth year. Instances have, however, been observed up to the fiftieth or even the sixtieth year. During the first year of life also it is rare. (2) *Sex*.—This is without appreciable influence. (3) *Season*.—Cases are more numerous in winter and spring than at other seasons. (4) *Climate*.—Diphtheria is met with less frequently in tropical than in temperate and cold climates. Humidity favors the propagation of the diphtheria germ, and hence damp cellars also promote the spread of the disease. (5) *Unhygienic Conditions*.—Unfavorable sanitary surroundings tend to lower vitality, and in consequence to increase the susceptibility to the specific virus. Most epidemic outbreaks have held more or less intimate relationship with decomposing organic matter, defective drainage and sewage, cesspools, etc., though it is to be especially remembered that the disease often prevails in sparsely-settled rural districts.

**Immunity.**—A single attack does not confer perfect immunity. Second and third attacks not infrequently occur in the same individual.

**Symptoms.**—**Incubation.**—The duration of this period is from two to seven or ten days, and in a small percentage of the cases it may be longer. In virulent epidemics and when the disease is produced experimentally the incubation-stage is short—from twelve hours to two or three days. The prodromal indications of diphtheria are not strikingly characteristic. They may either be acute in character or very mild; but usually the child will complain of feeling weary and indisposed to play, of being chilly and cold, and of pain in the head, back, and limbs. There is nothing in this early stage of the disease to distinguish it from many of the other affections of children, such as simple pharyngitis or tonsillitis. There may be some fever, not very high, and an elevation of one or two degrees at most. The child may often complain of discomfort in swallowing, and on examination the fauces will be found to be reddened, and in a short time the exudate will be found on the tonsils or soft palate. This is the usual type of **simple tonsillar diphtheria**.

**Pharyngeal Diphtheria.**—The symptoms are usually slower of development than in tonsillitis. The child is sluggish, looks heavy-eyed, languid, and pale for several days. The fever may not rise above 101° or 102° F. (38.8° C.). On examining the throat, however, it is found



to be swollen and red, and if lividity is more pronounced than the swelling, it suggests the true nature of the disease. The membrane begins on the tonsils in the form of small patches of yellow exudate, scarcely distinguishable from the thick, cheesy plugs of inspissated dead epithelium and secretion which issue from the mouths of the follicles of the tonsils during the course of acute or chronic tonsillitis. The membrane spreads from the tonsils to the soft palate and half arches within a few days, and it may also appear on the pharyngeal wall. During this stage the throat may become much swollen and the tonsils greatly enlarged, frequently meeting in the median line. The glands immediately beneath the angle of the lower jaw on one or usually both sides become hard, painful, and slightly enlarged; the swelling of these glands is not usually great in mild forms, although their presence, in association with the foregoing symptoms, is an infallible indication of the disease. The child, as a rule, shows grave constitutional symptoms for a few days, and loses its appetite. The temperature is not characteristic, as a rule not being high, and the pulse is rapid and weak, being out of proportion to the general indications of the disease. In mild cases the symptoms abate by the end of the first week, and the pseudo-membrane separates, leaving a red, inflamed surface behind. The child is prostrated for a number of weeks, and in about 20 per cent. of all mild cases the toxic effects of the disease may show themselves in the form of a neuritis, with its accompanying paralysis.

*Variations in Manifestation.*—Diphtheria may exhibit a number of variations as regards the seat of attack and the severity of the poisoning. In some epidemics the Klebs-Löffler bacillus seems to be more active and more numerous, or perhaps more virulent, than in others. The severity of the attack does not seem to depend on the amount of the pseudo-membrane, but rather, according to Rotch, upon three factors: (1) the virulence of the bacteria; (2) the local resistance; and (3) the general resistance. The false membrane is most frequently seen on the tonsils, spreading gradually to the soft palate and uvula, though the mucous membrane of any part of the body may be the seat of the growth.

*Malignant Diphtheria.*—The symptoms are severe from the commencement. There are one or at most two days of slight illness, and then alarming symptoms manifest themselves, cardiac failure possibly setting in without a specially severe local lesion. Vomiting and high fever, resembling the onset of scarlet fever, may initiate the attack; and within a few hours we may find extensive swelling at the angles of the jaws, with a feeling of stony hardness, a very offensive, bloody discharge coming from the nostrils, accompanied with difficulty in opening the mouth. If the throat is examined, there will be found extensive swelling of the tonsils, even to meeting, the uvula and soft palate being edematous and covered with much sloughy-looking membrane. The temperature in severe cases soon reaches a point between 103° and 104° F. (40° C.), while the heart-beats become exceeding feeble. In a day or two the cellulitis extends, the face becomes edematous, the skin pits all over the face, neck, sternum, and chest-walls. The patient soon becomes drowsy, cyanotic, and occasionally an erythematous rash appears about the face, neck, and chest, while a purpuric rash is not in-



frequent in malignant cases. Death occurs in such cases within one week from toxic poisoning. Malignant cases of diphtheria resemble very closely malignant scarlet fever, though the pulse in scarlet fever will be of assistance in the absence of the characteristic rash.

*Nasal Diphtheria.*—In all severe cases of pharyngeal diphtheria the inflammatory process is likely to extend to the nasal mucous membrane. In some cases the nasal mucous membrane is found to be the first involved, and it may spread to the tonsils, but in these cases the exudate will be found to involve the back of the soft palate and pharynx as well. In many cases of nasal diphtheria no membrane may be found during life; there may be only a purulent discharge with blood, the presence of which in the nasal passage obstructs breathing, giving rise to a bubbling sound, and rendering sleep troublesome and noisy. Many cases have also been reported of formation of pseudo-membrane in the nose with mild general symptoms (often insignificant), and from which bacilli identical with diphtheria bacilli were obtained by culture, the bacilli often persisting for months. Sometimes the cases have recurring mild attacks of pseudo-membranous inflammation of the nose, while the bacilli may be constantly present. It is probable that these cases may give rise to infections of like nature, and even of true diphtheria. In nasal diphtheria the symptoms are quite as severe as in faucial diphtheria, and in cases in which the soft palate, tonsils, and nasal mucous membrane are involved the general symptoms, the depression, and also the albuminuria, are well marked. In this place it is well to remember that in measles we sometimes have a form of membranous exudation occurring on the nasal mucous membrane and as a primary disease which is not diphtheria. This disorder runs a more favorable course, the membrane being thinner and less adherent, than in diphtheria. “Rhinitis fibrinosis” is of favorable prognosis. In all cases of coryza with fever we should be guarded as to opinion, especially if an epidemic of diphtheria is prevalent at the time. The diphtheritic inflammation may spread from the nose to the conjunctivæ, with the formation of a false membrane, and much purulent discharge may escape from the eyes, the lids of which may be greatly swollen.

*Wound-diphtheria.*—A diphtheritic membrane may grow on the lips, tongue, vulva, or glans penis. The bacillus will not live on normal skin, but when the skin is cut or bruised, as after blistering or an eczematous condition, and when a moist, raw surface is present, the bacillus freely flourishes. Granulations also form a favorable soil. The diphtheritic germs may be introduced into the system during an operation, such as an excision of the tonsils, or even a vaginal examination; and in new-born infants the granulating surface left after sloughing of the cord may become the seat of diphtheritic inflammation.

*Laryngeal Diphtheria or Membranous Croup.*—In many cases the Klebs-Löffler bacillus produces its influence first on the mucous membrane of the larynx, and in these cases the mucous membrane of the nose and pharynx may never give evidence of a false membrane. In laryngeal cases the first symptom is a cough of a harsh, metallic, ringing character, and never to be forgotten when once heard. The temperature may be slightly above normal, or even, in many cases, normal. The toxic absorption is slight, on account of the locality affected, and the

constitutional symptoms are usually mild. The local symptoms, however, are very alarming, as they are the results of laryngeal obstruction, there being marked dyspnea with retraction of the intercostal and supraclavicular spaces, and later of the epigastrium and lower chest. These are associated with an increasing cyanosis. The child is soon very restless, is forced to sit up to breathe, and for the same reason bends forward with its head thrown back. In these extreme cases, unless relief is soon gained, the child dies of suffocation. In many cases the slower form of suffocation may result from the extension of the membrane downward to the bronchi.

**Complications.**—Local complications may be mentioned, as when we have *hemorrhage* from the nose and throat in the more severe ulcerative cases. *Skin-rashes* are not unusual, especially the *diffuse erythema*. Sometimes *urticaria* will be noticed, and in very severe cases *purpura* will mark the skin.

*Broncho-pneumonia* is the most serious pulmonary complication of diphtheria. It is not produced by the Klebs-Löffler bacillus, but by pyogenic cocci which have been taken in during respiration. Broncho-pneumonia is very frequent, and most usually terminates laryngeal cases that have been operated upon.

*Albuminuria* is really a part of the disease, and can scarcely be regarded as a complication. It is the most constant symptom, and is almost as certain in establishing a diagnosis of true diphtheria as a bacteriologic examination. It is met with in both mild and very severe cases, and the greater the amount of albumin the more severe the case. When acute nephritis complicates diphtheria it is usually not accompanied by edema or anasarca.

*Dysphagia* may, by its constant existence throughout the disease, produce a profound impression on the general nutrition. Involvement of the *conjunctivæ* is a rare and very grave complication.

*Otitis media* occurs frequently, and may be a very troublesome complication as well as a sequela.

The most frequent *sequelæ* are anemia, chronic naso-pharyngeal catarrh, peripheral neuritis, and its associate paralysis.

*Anemia* may so prolong convalescence that the child will frequently be exposed to some intercurrent disorder. The *chronic naso-pharyngeal catarrh* may be so marked as to offer a favorable ground for new diphtheritic invasion. *Neuritis* and *paralysis* will not be noticed until the third or fourth week, the paralysis usually being first seen when the child attempts to swallow and the food is regurgitated through the nose. This is due to a paralysis of the muscles of the soft palate, which will also be noticeable owing to a peculiar alteration of the voice. The paralysis may take a general form, such as is seen in multiple neuritis, the lower extremities being affected and the knee-jerks absent. The paralysis is frequently quite extensive; it may extend to the external ocular muscles and cause squint, to the ciliary muscles and cause dimness of vision from unequal accommodation, or to the muscles of the trunk in general, producing a very general paralysis. The child, not being able to hold anything, may stagger about as if intoxicated, so much so as to suggest the existence of a cerebral tumor. The disturbance of vision and the absence of the patellar tendon reflex has in adults led to a mistaken diag-



nosis of *locomotor ataxia*. Loss of taste, deafness, and a disturbance of sensation are not infrequent. Thus, paralysis is to diphtheria what dropsy is to scarlet fever—a proof positive of the disease. To make one step more, in many sudden deaths occurring in early diphtheria we must recognize paralysis of the heart outside of all toxic influence, and the fact that in cases of sudden death, which are by no means uncommon during the disease, we have some sudden disturbance of the vagus brought about by means of its cardiac branches.

The prognosis in all cases of post-diphtheritic paralysis is very favorable. Myocardial weakness tends to supervene as a sequel. It is evidenced by the sudden accession of pallor, nausea, sometimes by vomiting, and also by weak heart-sounds and a feeble, broken, irregular pulse, etc.

**Diagnosis.**—The diagnosis of a pharyngeal diphtheria (the usual typical form) is not difficult if an epidemic be prevailing. The false membrane on the fauces and the presence of albumin in the urine give us a practically certain diagnosis. From *follicular tonsillitis* we differentiate diphtheria by the seat of the membrane, that of the former being *in* the tonsils, while diphtheritic membrane is *over* the tonsils and *over* the soft palate. Moreover, in follicular tonsillitis the fever is high, the onset is sudden, and it is most usually associated with gastric disturbance. Albuminuria is generally present in diphtheria, while it is present in follicular tonsillitis in exceptional cases only. The histories of the two cases are quite different. In many cases clinical distinctions will entirely fail us, it being uncertain whether or not the case is one of mild diphtheria, and then the most certain evidence of the disease is the finding of the Klebs-Löffler bacillus in the membrane. In many cases of so-called diphtheria the membrane is only formed by streptococci or staphylococci.

The *croupous* or *membranous angina* may offer some doubt, yet in this disease there is no tendency to spread to the nasal mucous membrane or to the larynx; there is a diminution in the glandular enlargement; there is no albumin and the onset is more sudden.

A mild case of diphtheria in a house may be followed by a malignant one. Moreover, mild cases may at first not contain albumin, and fail to show its presence until later in the disease. Diphtheria frequently is associated with a rash, rendering it difficult to distinguish the condition from scarlet fever; but in diphtheria the rash is erythematous in character, while in scarlet fever the rash may be absent. The glandular swelling and sloughy condition of the throat, however, closely resemble diphtheria, and a positive diagnosis without a bacteriologic examination is often impossible.

An immediate diagnosis without the use of culture is often possible by making a smear-preparation of the exudate from the throat (see Fig. 19), when the Klebs-Löffler bacilli may be present in sufficient numbers, and may be quite characteristic to an expert. In this connection may be given the following statement by Park, who has had an exceptional experience: "The examination by a competent bacteriologist of the bacterial growth in the blood-serum tube, which has been properly inoculated and kept fourteen hours at the body-temperature, can be thoroughly relied upon in cases in which there is a visible membrane in the throat if the culture is



made during the period in which the membrane is forming, and no antiseptic, especially no mercurial solution, has lately been applied. In cases in which the disease is confined to the larynx or bronchi, surprisingly accurate results can be obtained from cultures, and although, in a certain

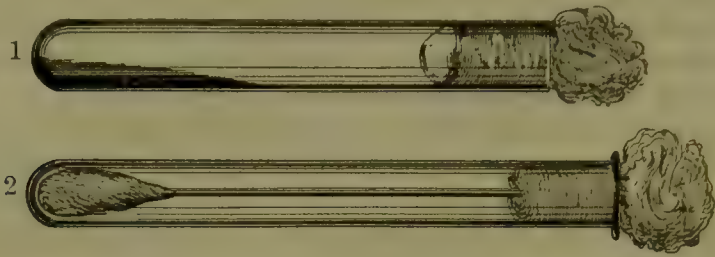


FIG. 19.—1, A tube of blood-serum; 2, a sterilized cotton swab in test-tube.

Rub the swab gently but freely against the visible exudate, and without laying it down, after withdrawing the cotton plug from the culture-tube, insert it into the latter, and rub that portion which has touched the exudate gently but thoroughly over the surface of the blood-serum without breaking its surface. Now replace the swab in its own tube, plug both tubes, and place them in the box provided by the health officials. This is to be sent to the bacteriologic expert. In laryngeal diphtheria the swab is to be passed far back and rubbed freely against the mucous membrane of the pharynx and tonsils.

proportion of cases, no diphtheria bacilli will be found in the first, yet they will be abundantly present in later cultures. We believe, therefore, that absolute reliance for a diagnosis cannot be placed upon a single culture from the pharynx in purely laryngeal cases." When a bacteriologic examination cannot be made the practitioner must regard as suspicious all forms of throat-affections in children, and carry out measures of isolation and disinfection. In this way alone can serious errors be avoided. Mistakes do not usually occur in a more pronounced membranous sore throat, but in the lighter types, many of which are in truth due to the Klebs-Löffler bacillus.

**Prognosis.**—Diphtheria is at the same time the most prevalent and most fatal of all the diseases with which the general practitioner has to deal, and, I may add, the least understood. The mortality is enormous, though it differs widely in different epidemics, and the most fatal variety is unquestionably the laryngeal. In laryngeal diphtheria the mortality may be as high as 75 per cent., and the younger the child the more unfavorable the prognosis, the strong and healthy seeming to share the same fate as the weakly. Of especially unfavorable prognosis are those cases that show large quantities of albumin in the urine, general adenitis, cervical glandular enlargement, excessive nasal discharge, a necrotic state of the throat, vomiting, and partial or complete suppression of the urine. Although the temperature in diphtheria is never very high, yet a sudden fall of temperature to subnormal and an irregular pulse are also very unfavorable symptoms. Recovery from a severe attack in which there is extreme depression and much albumin is unusual, especially in a child under six years of age, though recovery takes place very frequently in what would be regarded as hopeless cases. Suppression of urine, if it continues thirty-six hours, is generally fatal. A fall of temperature in scarlet fever, if we have a strongly-acting kidney, is an encouraging indication; the same, however, is not true in diphtheria if it is associated with vomiting. The results of Morse's extensive observations are opposed to those of Bouchut and Dulinsay, who claim that the degree of leukocytosis is of prognostic value (see p. 180). The cases of neuritis invariably

recover. A child who has had diphtheria once is most likely to contract it again, and if he recovers is liable to suffer from its effects for years.

*The causes of death in diphtheria*, in their order, are as follows: involvement of the larynx; membranous croup or laryngeal diphtheria; septic infection, which may be a slow death; sudden heart-failure—paralysis of the heart; broncho-pneumonia, following tracheotomy or occurring during convalescence.

**Treatment.**—**Prophylaxis.**—The best preventive measures against diphtheria are a clean nose and mouth. Insist upon a careful toilet of the nose in all children. The slightest appearance of a coryza must be overcome at once by the use of a mild antiseptic wash; all accumulations of crusts, dust, dried blood, etc. should be removed from the nose twice daily, especially in children attending school or during the prevalence of an epidemic. The child should be early taught to employ a small antiseptic gargle as a daily routine, using a weak solution of hydrogen dioxid, listerin, or even a mild dilution of alcohol. The teeth should be carefully cleaned daily, and all decaying teeth should be filled or removed. If it is true, as one authority claims, that over two hundred different species of bacteria find a happy home in the oral cavity, this fact should make all parents attentive to the proper physiologic condition of the mouths of their children.

All cases of sore throat should be examined for the Klebs-Löffler bacillus, and, if it is found, the individual should be isolated; and all cases of diphtheria should be kept isolated until the membrane has disappeared from the nose and throat. This is especially true in schools and asylums. Moreover, the throats of all persons exposed to this disease, and of those caring for diphtheritic patients, should be frequently examined for the Klebs-Löffler bacillus, and if it be found the person should receive immunizing doses of antitoxin. The fact that the Klebs-Löffler bacilli when found in healthy throats may not be active is no argument against isolation, because it is well known that if the same germs were to find such favorable soil as a broken or catarrhal membrane they would rapidly develop. The seed being there, the soil only requires preparation for its reception.

An unrecognized feature in the prophylactic treatment of the disease is seen in the uncertain period of convalescence. It has frequently happened that long after all membrane has disappeared active bacilli may still cling to the throat. This condition may continue from two to six months, and even longer in deeply fissured tonsils; and the disease may be communicated by such throats in the act of kissing young children or adults with sensitive throats or with a broken mucous membrane of the mouth. For this reason the indiscriminate kissing of young children on the lips should be interdicted by the physician.

Sufficient importance has not been given to the milder cases of diphtheria as to their isolation and disinfection, and this fact explains the occurrence of many house-epidemics.

**Treatment of the Attack.**—The treatment falls very naturally under several departments: (*a*) the hygienic measures to limit the diffusion of the disease; (*b*) the local management of the throat to destroy early the toxic germs; (*c*) medication to antagonize the effect of the toxins, and eventually to overcome the complications and sequelæ.



(a) **Hygienic Treatment.**—The patient should be in a room well exposed to sunlight and fresh air, as diphtheritic germs grow well in poorly-lighted and damp chambers. No stationary washstand should be allowed in the room, and Goodhart well says that many cases seem to have their origin in the proximity to foul-smelling drains. The physician should never consent to be responsible for the recovery of a patient in a room in which there is a washstand with its uncertain connection with the main sewer. If possible, the patient should use two connecting rooms, one during the day and the other at night, so that one while not in use may be thoroughly aired and disinfected. Even in mild cases the patient should be kept in bed throughout the attack, and in more severe cases also for some time during convalescence. This is especially important when there have been symptoms of cardiac depression during the acute stage. The general comfort of the patient is enhanced by two daily sponge baths of tepid salt-water or of alcohol and water.

**Feeding.**—Nursing infants may be fed on breast-milk obtained by a breast-pump, but should not be placed at the mother's breast (Holt). The feedings should be regular, yet lighter in quality and quantity than in health, remembering the tendency to vomit in all acute febrile affections, and the fact that gastric disturbance is closely associated with diphtheria. The rule must be, less solids and more fluids than in health. Milk in some form being our main dependence, it should usually be diluted, and for young children partially if not wholly peptonized. The greatest difficulty comes in the latter part of the disease, when the child is septic and most likely has a strong objection to be disturbed. At this time vomiting is most easily provoked, and swallowing is rendered very difficult on account of the swelling and pain. We must not neglect the feeding even if it does cause discomfort, and here forced feeding by means of gavage is most valuable. Gavage is more desirable and likely to be more successful with children under three years than rectal alimentation. In older children, who object to the tube through the mouth, it may be passed through the nose with very little difficulty, and gavage by this route, even in intubated cases, will be extremely satisfactory. Concentrated broths, meat-juice, and even milk-punch or raw eggs, may be given in this way.

**Stimulants.**—Alcohol no longer holds a debatable ground in the treatment of diphtheria: it is the most powerful drug in our possession to offset the ravages of the disease on the nervous centers and for the control of the circulation. Stimulation should be commenced as soon as there is a reasonable certainty as to the correctness of the diagnosis, and by commencing early with whiskey or brandy we may prevent the depressing effects of the poison of diphtheria as seen in the pulse and general condition of the child. The indications for alcohol are marked prostration, feeble pulse, and a weak first sound of the heart. The quantity must be adjusted to the age and gastric condition of the child, and usually one ounce (32.0) of good whiskey or brandy, well diluted, in twenty-four hours is sufficient for a child four years old. In very bad cases five or six times this quantity may be given, the only limit being the tolerance of the stomach. As a rule, the stimulant should be mingled with the food, as the child may rebel against taking both food and stimulant.

Strychnin stands next to alcohol in importance in the treatment of



diphtheria, and usually it is given in too small doses. For a child four years old gr.  $\frac{1}{30}$  (0.0021) may be given every six to eight hours, and may be administered in little tablets by the mouth or hypodermically.

Digitalis does not hold an important place in the heart-weakness of diphtheria, and yet it is strongly indicated on theoretic grounds. Clinically, it has been found to have an unfavorable action on the stomach before its good influence can be had on the heart itself. The same may be said of camphor and ammonium carbonate. The aromatic spirits of ammonia is valuable for rapid effects in syncopal attacks. In cases of threatened heart-paralysis occurring late in the disease Holt has found nothing so valuable as morphin employed hypodermically, the drug being given in full doses and repeated every two hours, keeping the child under its influence for some days.

Internal medication should be avoided until absolutely necessary, and such symptoms as vomiting or diarrhea are to be met with sufficient treatment only for their control.

(b) **Local Treatment.**—For the direct attack upon the membrane in the throat nearly all the remedies of the Pharmacopeia have been used. Gargling, swabbing, painting, spraying, and washing the throat out, all have their advocates, and every physician has his favorite remedy or combination. And, as all adult pharyngeal diphtheria tends to recovery, it would seem reasonable that this form of treatment should not be neglected; yet since the acceptance of the antitoxin treatment medical opinion has suffered a decided change, especially as to the importance of local measures. The very best local application for pharyngeal or nasal diphtheria consists of hydrogen dioxid, diluted one-sixth, and used both as a gargle and spray as most convenient; this is usually sufficient in the early stage. The tincture of iron and glycerin is a valuable local remedy applied by means of a swab. The object of local treatment in the light of our new pathology is a more thorough cleanliness, and not the destruction of the bacilli, yet it does still more good by preventing the systemic absorption of the ptomains. Hence a careful toilet of the nose and throat is important in preventing the spread of the disease. This part of the work is more easily directed than accomplished, especially in rebellious children, and we have frequently felt that new lesions were created in the mucous membrane of the nose and throat by an undue ardor in making applications. To avoid new lesions the spray alone should be used, and for the nose boric-acid solutions or hydrogen dioxid, 1:10, will be most serviceable. In this work the utmost tact and kindness must be maintained, for it is truly pitiable to force a struggling child, endangering the strength to accomplish so little. Warm, weak solutions, most thoroughly applied by means of the fountain syringe, will be better than the more frequent use of the hand-syringe. In older children who will use it a gargle of boric acid, listerin, or Dobell's solution, well diluted, may be used to keep the nose and mouth clean.

In laryngeal diphtheria the child should inhale an atmosphere laden with the vapor of slaking lime, or, whenever practicable, an atmosphere saturated with Löffler's solution (menthol 10 grams, dissolved in sufficient toluol to make 36 c.c., liq. ferri sesquichlorid, 4 c.c., absolute alcohol, 60 c.c.). The development of the signs of actual stenosis, as shown by stridulous breathing, cyanosis, etc., furnishes an indication for either in-

tubation or tracheotomy. According to my own observations, the results of intubation have been quite favorable, and I would strongly recommend

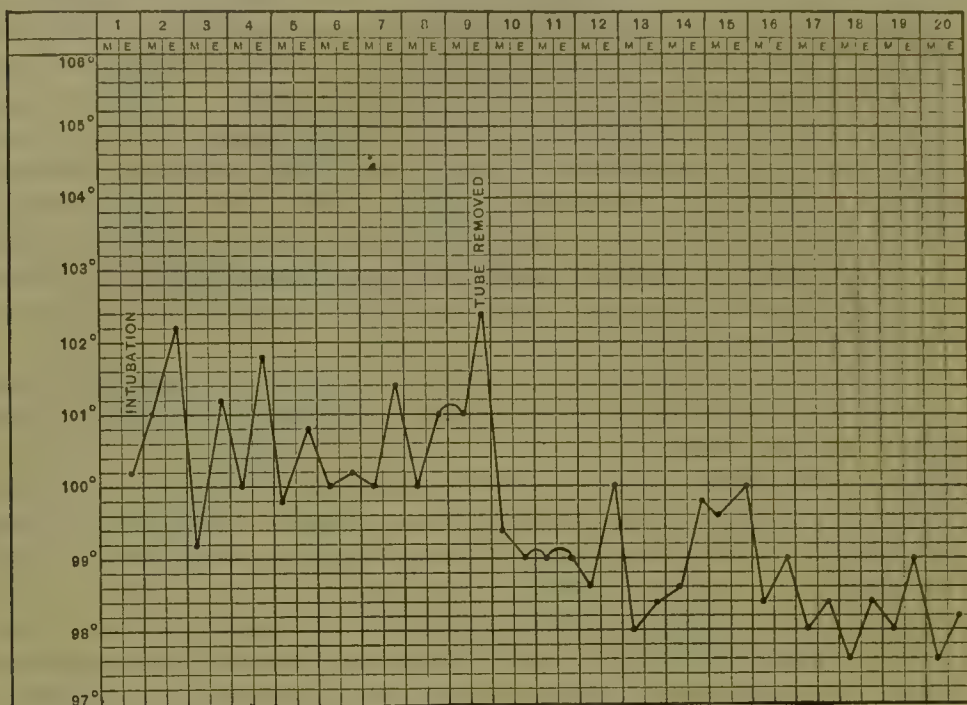


FIG. 20.—Temperature-chart of a case of diphtheria.

a trial of this procedure before resorting to tracheotomy (see temperature-chart, Fig. 20).

(c) **External Applications.**—External applications to the throat have no effect on the course of the disease. They are useful, however, in relieving the pain and the swelling in the lymph-glands. Careful massage of the neck with camphorated oil, as hot as the skin will tolerate, is very soothing; and soap liniment may be used in the same way, or if much pain exists chloroform liniment may be substituted. Poulticing for the relief of pain is not desirable, as it seems to favor suppuration. In older children the ice-bag has been used with good effect, and it soon brings grateful relief from the tension and subdues inflammation. All manipulations about the child, however, should be carried on as gently as possible, so that its rest may not be disturbed.

**Serum-therapy; the Antitoxin Treatment.**—This has now passed beyond the stage of uncertainty and experimentation, and must be regarded as one of the most positive advances made in practical medicine. Its utility rests upon the discovery that animals may be rendered immune to diphtheria, and that the blood of an animal so treated, when introduced into another animal, protects the latter from infection by the diphtheria bacilli. The studies of Behring, Roux, Kitasato, and others have demonstrated that the use of the blood-serum of the lower animals, artificially rendered immune against diphtheria, has a powerful healing influence upon diphtheria that has been contagiously or spontaneously acquired by man. These experiments were first published in December, 1890. The principle was first shown to be true of tetanus, and, late in 1892, Behring further showed that the blood of an immunized animal had the power



both of protecting and of curing susceptible animals which had been inoculated either with the toxins or the bacilli of diphtheria. In preparing the blood-serum it is very desirable, of course, to have a uniform strength or standard. One-tenth of one cubic centimeter of what Behring calls his normal serum will counteract ten times the minimum of diphtheria poison, fatal for a guinea-pig weighing three hundred grams. One cubic centimeter of this normal serum he calls an antitoxin unit. The serum prepared by his method is labelled in three strengths. No. I. is sixty times the strength of the normal serum; No. II. is one hundred times as strong; and No. III. is one hundred and forty times as strong. To a child of two years or over not less than 800 or 1000 units should be administered at the first dose; hence solution No. I. is rarely employed at the present day. Should a favorable result not be attained, then, on the following day, 1500 to 2000 units should be administered, and a third dose after a similar interval if necessary. The latter dose should be employed at the outset in very severe cases and in those not seen until they are far advanced. The sites to be selected for injection are various. In very young children either the buttock or thigh is to be preferred, while in older children the flanks or subscapular spaces may be chosen as well. The injections should be made deeply into the subcutaneous cellular tissue.

In fortunate cases the influence of the serum soon becomes apparent. Within twenty-four hours the faucial swelling diminishes, the membrane exfoliates, the temperature falls, the pulse becomes slower and stronger, and the general condition of the patient quickly improves. In cases of moderate severity and when injections are employed early the improvement in the throat and the constitutional symptoms is very decided; and the earlier the case comes under treatment the better are the results. There are, however, many cases of great severity in which the antitoxin has been used early, and yet has not shown any benefit.

A danger in serum-therapy may be the development of local abscesses, which, if full antiseptic precautions be taken, must be rare indeed. I have escaped them altogether. Certain skin-eruptions have been observed after injections, mostly urticarial, though sometimes scarlatiniform. The latter form has given rise to apprehensions of scarlatina. Widerhofer had one case which was isolated as measles, but never developed any symptoms other than the suggestive eruption. Rarely, joint-pains and swellings, with general prostration, supervene. Two fatal cases have been reported—one<sup>1</sup> that of a healthy boy five years old, the result of an injection of Behring's fresh serum as a preventive, dying within five minutes; the other occurred in Berlin.<sup>2</sup>

For establishing immunity in subjects exposed to infection the injection of 60 units (1 cubic centimeter of the No. I. serum) affords protection. In order to arrest the development of the disease during the period of incubation 100 units (1 cubic centimeter of No. II. serum) is probably sufficient.

A large number of preparations are on the market, many of which are good, yet great caution must be exercised in their selection.

The use and value of antitoxin in private practice are best shown in

<sup>1</sup> *Journal of the American Medical Association*, April 4, 1896.

<sup>2</sup> *Medical News*, April 18, 1896.



the following summary of the report of the American Pediatric Society's investigation of the subject:

1. The report includes returns from 615 physicians. Of this number more than 600 have pronounced themselves as strongly in favor of the serum-treatment, the great majority being enthusiastic in their advocacy.

2. The cases included have been drawn from localities widely separated from each other, so that any peculiarity of local conditions to which the favorable reports might be ascribed must be excluded.

3. The report includes the record of every case returned, except those in which the evidence of diphtheria was clearly questionable. It will be noted that doubtful cases that recovered have been excluded, while doubtful cases that were fatal have been included.

4. No new cases of sudden death immediately after injection have been returned.

5. The number of cases injected reasonably early, and in which the serum appeared not to influence the progress of the disease, was but 19, these being made up of 9 cases of somewhat doubtful diagnosis, 4 cases of diphtheria complicating measles, and 3 malignant cases in which the progress was so rapid that they had passed beyond any reasonable prospect of recovery before the serum was used. In 2 of these the serum was of uncertain strength and of doubtful value.

6. The number of cases in which the patients appeared to have been made worse by serum was 3, and among these there is only 1 case in which the result may be fairly attributed to the injection.

7. The general mortality in the 5794 cases reported was 12.3 per cent., and, excluding all cases moribund at the time of the injection or dying within twenty-four hours, it was 8.8 per cent.

8. The most striking improvement was seen in cases that were injected during the first three days. Of 4120 such cases the mortality was 7.3 per cent., and, excluding cases moribund at the time of the injection or dying within twenty-four hours, it was 4.8 per cent.

9. The mortality in 1448 cases injected on or after the fourth day was 27 per cent.

10. The most convincing argument, and, to the minds of the committee, an absolutely unanswerable one, in favor of serum-therapy is found in the results obtained in the 1256 laryngeal cases (membranous croup). In one-half of these, in a large proportion of which the symptoms were severe, recovery took place without operation. Among the 533 in which intubation was performed the mortality was 25.9 per cent., or less than half as great as has ever been reported by any other form of treatment.

11. The proportion of cases of broncho-pneumonia (5.9 per cent.) is very small, and in striking contrast to results published from hospital sources.

12. As against the two or three instances in which the serum is believed to have acted unfavorably upon the heart might be cited a large number in which there was a distinct improvement in the heart's action after the serum was injected.

13. There is very little, if any, evidence to show that nephritis was caused in any case by the injection of serum. The number of cases of

genuine nephritis is remarkably small, the deaths from that source numbering but 15.

14. The effect of the serum on the nervous system is less marked than upon any other part of the body; paralytic sequelæ being recorded in 9.7 per cent. of the cases, the reports going to show that the protection offered by the serum is not great unless injections are made early.

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## SEPTICEMIA.

**Definition.**—A disease due to an introduction into the system of the products of putrefaction (sapremia) or to a microbic invasion of the blood and tissues (true septicemia), with or without the presence of a local seat of infection.

**Pathology.**—After death the body putrefies early. The macroscopic changes in the viscera are sometimes few and often wanting. The muscles present a brownish color-tint. The pia mater is generally congested, and, together with the nerve-centers, may be the seat of ecchymoses. The blood is dark (“tar-like”); its coagulability is diminished, and, microscopically, it shows an abundance of micrococci and bacilli. The spleen is somewhat softened and its lymphoid elements more distinct, and almost invariably ecchymoses are found in the serous membranes, especially the pericardium and peritoneum.

In protracted septicemia more marked alterations exist, and among them may be briefly enumerated the following: endocarditis (rarely ulcerative); gastro-intestinal catarrh (of the duodenum and rectum in particular) with punctiform extravasations; enlargement of the lymphatics and spleen, with softening of the latter; cloudy swelling of the liver (rarely the so-called emphysema of the organ due to putrefaction); edema and catarrhal inflammation of the uriniferous tubules; congestion, sometimes associated with edema of the lungs; and inflammation of the pleura, pericardium, and peritoneum, with ecchymoses and trivial effusions.

Microscopically, the internal organs show numerous small foci of inflammation, some of which may be the seat of “coagulation-necrosis.” Bacteria are found in abundance in various situations, such as the exudations, the capillaries of the inflammatory foci, and especially in the renal glomeruli.

**Etiology.**—**Bacteriology.**—Septicemia is due to micrococci which Koch has shown to be considerably smaller than pus-cocci, though no one form of bacterium has been found constantly present to the exclusion of all others. Besser, as the result of careful experiments, concludes that septicemia is caused solely by streptococci, while Rosenbach and others have found both staphylococci and streptococci. Doubtless in many instances of human septicemia the clinical manifestations are due partly to bacterial poisoning and partly to septic intoxication with the poisons (ptomains) developed by the organisms, and the ptomains probably kill the patient before the bacteria can propagate themselves throughout the system. Laboratory experiments teach us that in the

lower animals septicemia can be produced both by chemical poisons and by bacterial infection, and these two types are observed in human beings. With reference to the bacterial form Warren<sup>1</sup> states: "Whether this process is caused solely by the multiplication of bacteria, or is dependent in part upon the liberation of intensely powerful poisons, or is due to some ferment-like substance capable of reproducing itself like the poison of the serpent, as are diphtheria and tetanus, much more extensive studies upon the human subject will be necessary to enable us to say."

**Modes of Infection and Introduction of the Poison into the System.**—(1) **Wounds**, either surgical or the result of injury, with which we have nothing further to do in this work. Since the days of rigid antiseptic precautions this mode of entrance is, comparatively speaking, uncommon.

(2) **Through the uterus**, following labor, miscarriage, or abortion. Generally in these cases there are accompanying local changes, but in a few the poison appears to pass the unguarded portals of the organ, while the latter exhibits nothing abnormal.

(3) The cases in which the poison gains entrance into the body **without obvious wounds** or raw surfaces are relatively more common. When the skin is quite natural, septic infection or intoxication cannot occur, but the slightest abrasion or cut, bed-sore, etc. may serve as a gate of admission. These slight lesions "may be almost completely healed by the time the severe symptoms of the disease are developed" (Strümpell).

(4) **Mucous membranes** often admit the virus, being less protective in nature than the skin. The numerous bacteria—benign and pathogenic—that are constantly present in the intestinal canal may also find in local lesions (as in typhoid fever, dysentery, etc.), or catarrhal inflammation even, points of lodgement and cause a systemic infection. To this category belongs that form of septic infection which follows gonorrhea. The so-called cases of "spontaneous septicemia" are also usually occasioned by absorption from the mucous surfaces.

Rheumatic or septic manifestations often follow attacks of *tonsillitis*, and it is probable that the tonsils are more frequently points of entrance for the organism than has hitherto been supposed (Wade, Banatyne).

(5) "**Sepsis Intestinalis**."—This special form of poisoning is caused by canned meats, ice cream, sausages, and cheese. Vaughan, to whom we are indebted for the first description of "sepsis intestinalis," found in cheese a ptomain which he named *tyrotoxicon*, and which he regarded as the active agent in this group of poisoning cases. The symptoms are due, according to his statement, to poisoning by chemical substances, being instances of *sapremia*; but it may yet be found that the intestinal micro-organisms play a more or less prominent part in the process.

(6) Ogston<sup>2</sup> recognizes as one of the mildest forms of sapremia the sickness and nausea produced by a bad smell, which, he claims, is but a ptomain of putridity that may, under certain contingencies, produce serious symptoms. On the other hand, persons who are habitually ex-

<sup>1</sup> *Surgical Pathology and Therapeutics*, p. 340.

<sup>2</sup> Warren, *loc. cit.*, p. 342.



posed to bad odors (workers in sewers, in the dissecting-room, etc.) may acquire a considerable degree of immunity against poisoning of this sort. The fever in these cases corresponds in severity to the dose of the poison.

(7) **Septicemia** may be associated with or follow osteomyelitis.

**Clinical History.**—(1) **Symptoms of Sapremia.**—The fact that this form may occur without bacterial infection, either local or general, must be emphasized, but more frequently there will be either local infection or putrefactive changes, with the production of a grave general condition due to the absorption of the poisonous chemical products. In certain other acute infectious diseases (diphtheria, tetanus, typhoid fever, erysipelas, etc.) the general symptoms are similarly engendered. Perhaps the most typical examples of sapremia seen by the physician are those due to tyrotoxicon and to the unaccustomed inhalation of foul odors. At the beginning a chill may occur, but this is more generally wanting. In "*sepsis intestinalis*" marked local symptoms may initiate the attack, as nausea, vomiting, colicky pains, diarrhea, etc., and in all forms there is fever, the temperature often rising rapidly to 101° or 103° F. (38.3°–39.4° C.) and sometimes higher. Prostration and anemia, particularly the latter, may be prominent symptoms. Microscopic examination of the blood generally shows leukocytosis, and always a marked reduction in the number of red corpuscles.

Sapremia following childbirth is a most typical sub-variety, and, apart from the special history, the symptoms are much the same as those above detailed. It is the form most amenable to treatment, the removal of the cause being followed by a rapid disappearance of all alarming symptoms.

(2) **Symptoms of True Septicemia.**—There is an incubation-period which is of variable duration, though usually averaging several days. The onset is more gradual than in the previous variety, and is rarely marked by a chill. Accession of fever following surgical procedures, with headache, anorexia, prostration, sometimes vomiting and diarrhea, and especially dulness occasionally amounting to mild stupor, announce the affection: these symptoms should also excite suspicion in the absence of obvious causal factors. They become intensified, and now the attack may closely simulate certain other infectious diseases (typhoid fever, acute miliary tuberculosis, ulcerative endocarditis, etc.), the clinical picture as outlined presenting nothing characteristic. There are, however, more or less distinctive features, which will be considered seriatim.

(a) **The Fever.**—This is usually of the *continued* type, and tends to increase in degree, fatal cases often terminating in hyperpyrexia. At the beginning the temperature may rise quite rapidly, and in some cases it may even be subnormal. Deep morning remissions may be observed.

(b) **The Circulatory System.**—The pulse is frequent, and near the end becomes very weak. In subacute cases characteristic lesions (endocarditis in particular) may develop, but are difficult of recognition, since they do not, as a rule, give rise to audible murmurs or other physical signs. In other instances soft murmurs may be heard, but it is indeed hard to discriminate these from functional sounds. Moderate leukocytosis is sometimes observed, and the presence of micrococci in the blood during life has been demonstrated.

(*c*) **Gastro-intestinal System.**—The spleen may become perceptibly enlarged, and gastro-enteritis is usually present, either in an acute form with vomiting and frequent serous discharges or more often merely with a diarrhea of moderate intensity (septic diarrhea).

(*d*) **Cutaneous Symptoms.**—Punctiform hemorrhages into the skin are of prime importance in the diagnosis. Occasionally more extensive ecchymoses appear, scarlatinal eruptions also showing themselves, but these are less characteristic. Among rare appearances herpes, roseola, edematous inflammations, and faint jaundice (affecting the skin and conjunctivæ) may be observed. The icterus is probably due to disintegration of the red blood-corpuscles in the liver.

(*e*) **Renal Symptoms.**—The lesions constitute the so-called "septic nephritis," the urine often containing a fair amount of albumin, epithelium, tube-casts, and red and white corpuscles.

**Diagnosis.**—(*a*) Sappremia can be distinguished by the history, the immediate appearance of the symptoms, their character, and by the prompt effect of the removal of the exciting cause. The diagnosis often requires a most careful search for the known etiologic factors, though even without the latter we can sometimes arrive at a correct conclusion by a careful process of exclusion.

(*b*) **True Septicemia.**—Here the existence of an incubation period, the continued fever, mental apathy, faint jaundice, splenic enlargement, and the characteristics of septic nephritis, all combine to form a well-defined group of symptoms. A careful blood-examination should be made for micrococci, etc., and cultures should be undertaken in spontaneous septicemia and other doubtful examples of the complaint. The surgeon should look to the condition of the wound if one is present.

**Course and Prognosis.**—The course may be brief, virulent attacks sometimes terminating fatally within forty-eight hours, this being especially true of sappremia when the dose of the poison is large. The gravity of the case in the latter form is in direct proportion to the amount of virus that enters the system, the outlook being good when the cause is removable. On the other hand, in true septicemia this avails nothing, the progression tending steadily to the end. The mildest types may, in rare instances, reach a favorable end, but the effects are not dependent upon the dose, and the minutest quantity may lead to specific results in their fullest intensity. It must not be forgotten that septicemia may pursue a chronic course in which the symptoms are milder, though the termination is very generally unfavorable, as in the acute variety.

**Treatment.**—Of first importance is the removal of the cause whenever practicable, this part of the treatment often falling within the domain of surgery. The physician must support the patient's strength by a suitable dietary and by the judicious use of cardiac stimulants: the former should consist mainly of liquids (milk, egg-white, meat-juice, etc.), and the latter of alcoholics, together with strychnin and ammonia. Of medicines, internal antiseptics (mercuric chlorid, creasote, etc.) richly deserve a trial, though striking results have not been obtained from their employment. The fever calls for antipyretics, such as quinin, phenacetin, acetanilid, together with hydrotherapy. Cardiac depressants, as acetanilid and phenacetin, should not be resorted to,

however, when great cardiac asthenia exists. To meet the renal condition the free use of water, together with the least irritating of the diuretics, is to be advised and encouraged. The other internal organs should also receive careful attention.

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## PYEMIA.

**Definition.**—A disease of the blood invariably associated with supuration, and due to an absorption of pyogenic organisms.

**Pathology.**—The cadaver does not undergo putrefaction as early as in septicemia. Briefly considered, the pathologic lesions that fall within the physician's province arrange themselves under the following heads:

(1) **Thrombosis and Embolism.**—At first the veins leading to and from the seat of the local changes from which pyemia arises contain thrombi which may soften into a puriform material. Thrombi are also found frequently in the lungs, a circulating embolus first finding lodgement in the pulmonary artery and its branches; they may be present in the liver, kidneys, spleen, cortical substance of the brain, and in other localities.

(2) **Abscesses.**—These so-called metastatic abscesses are set up by septic emboli or result from the thrombi (chiefly pulmonary and portal), and are found in the various internal organs, mainly, perhaps, in the lungs, liver, spleen, and kidneys. They are not large, but may coalesce and form cavities of the size of an apple. The kidneys are the chief organs of elimination in this disease, and hence it happens that numerous clumps of micrococci, producing miliary abscesses, are frequently seen in the regions of the Malpighian bodies. Infarction may be observed also. There are many other, though rarer, seats of abscesses, as the muscles, submucous and subcutaneous tissues, bones, the parotid gland, brain (cortical portion), ovaries, and testicles.

(3) **Lesions of the Skin and of Mucous and Serous Membranes.**—At the post-mortem examination hemorrhagic extravasations and pustules are often visible in the skin. The mucous membrane of the alimentary tract is rarely affected, differing in this point from septicemia, though occasionally ulcers may be noted, and most commonly in the stomach near the pyloric orifice (in puerperal cases) and in the large bowel. Probably they are always secondary to the submucous miliary abscesses. The serous membranes (pleura, pericardium, meninges of the brain, synovial membranes) may be the seat of purulent inflammation and of hemorrhagic extravasations.

(4) **Cardiac Lesions.**—Ulcerative endocarditis forms the chief morbid lesion. It begins in the form of small nodular vegetations upon the valves (most frequently the mitral), which disintegrate and leave ulcers behind (*vide* Ulcerative Endocarditis).

**Etiology.**—**Bacteriology.**—Experimental investigations have shown conclusively that the organisms usually responsible for this condition are the staphylococcus and the streptococcus. Whether the former or the latter be the agent of infection in the given case depends chiefly



upon the condition of the tissues at the starting-point, especially with reference to the character of the local defensive processes; also, though to a lesser extent, the degree of virulence of the micrococci.

Other important pyogenic micro-organisms are the gonococcus, pneumococcus, bacillus pyocyaneus, bacterium coli communis, bacillus tetragenus, and many of the specific micro-organisms.

**Paths of Infection of the Body.**—(a) Almost always the entrance is by *the blood-vessels*, the special varieties of micrococci that cause pyemia, reaching the veins and producing thrombo-phlebitis. Less frequently they reach the arteries and produce thrombo-arteritis. From the former condition emboli may be disseminated throughout the system, while from the latter the emboli are arrested in the neighboring capillaries to which the tributaries of the vessel lead. Micrococci independently of emboli may be found wandering in the blood-stream.

(b) Another path of entrance is *the lymphatic system*, but here the cocci meet with greater forces opposing their attempts to spread than in the blood-vessels, and hence it is a much rarer mode of propagation.

(c) In *spontaneous pyemia*, in which there is no wound to act as a point of departure, we must presuppose the existence of either a trivial lesion, as in “spontaneous septicemia,” or an area of lessened resistance. The latter may be produced by inflammation, by a contusion, and in other ways, and all that seems necessary is a lowering of the tone of the general system (Warren). I am certain that ulcerative endocarditis is not frequently the starting-point, but is usually secondary to foci of inflammation elsewhere, as claimed by Osler. The appendix is often the primary or original focus in this category of cases, micrococci localizing themselves here in consequence of a preceding disturbance of the circulation or catarrhal inflammation. I recollect one case in which no original abscess was found at the post-mortem.

**Predisposing Causes.**—(a) *Epidemic Influence.*—It has been proved by abundant experience that certain seasons are characterized by epidemic outbreaks of the disease.

(b) Cases have sometimes been noticeably more frequent in the early months of the year (February and March) than in other seasons.

(c) *Age and Sex.*—Males are more frequently affected than females, and most cases occur about the middle period of life or at the time of greatest danger from traumatism.

**Clinical History.**—**Incubation.**—The disease sets in from a week to ten days after the reception of the wound or even earlier, and always develops secondarily to suppuration somewhere in the body.

A most conspicuous symptom, and usually the first, is the *chill*: it may, however, be preceded for a variable time by fever of a continued or intermittent type. The fever of pyemia is of the suppurative type. Profound prostration develops early; the skin presents an icteroid appearance; and gastro-intestinal symptoms may appear, but are not prominent. The signs of abscess of the lung, liver, and other organs may develop in some cases, while in others the whole clinical picture is colored by the ill-defined characters of ulcerative endocarditis.

(a) **The Chill.**—This may be mild, though oftener it is quite severe. It is repeated at somewhat irregular intervals, and rarely it may recur

several times on the same day. Chills are most apt to occur during the daytime.

(b) **The Fever.**—A rapid rise of temperature accompanies the chill. The fever-curve is of the irregularly intermittent or profoundly remittent type, with intervening periods, showing slight or marked variations, and as decided deviations may occur within a short space of time, a two-hour record should be kept. The temperature rarely falls to the normal level; it may do so, however, and remain there for one or two days. To explain the peculiarities of the curve in this disease we need only recall the great variety of pathologic processes before noted. With the sharp fall of temperature *sweating* occurs, and leaves the patient more or less exhausted, though only temporarily so as a rule.

(c) **Respiratory System.**—Symptoms referable to the organs of respiration appear early. The pulmonary abscesses are usually latent, but may give rise to dyspnea, cough, and occasionally a purulent expectoration. Pain is present if they are superficially located, and under such circumstances the physical signs of cavity or of pleural effusion may be noted. The signs of pneumonia at one or both bases may also develop, the expectoration now becoming rusty.

(d) **Splenic and Hepatic Symptoms.**—The foci of suppuration in the liver are difficult of recognition unless they become large as the result of coalescence and are superficially located (see article Hepatic Abscess). Splenic infarction may also be safely diagnosed if there are pain and great tenderness (due to localized peritonitis) in the left hypochondrium, with progressive enlargement of the organ. In one case I detected distinctly crepitant sounds over the site of the spleen during life.

(e) **Cardio-vascular Symptoms.**—The pulse at first is accelerated, but moderately full and regular; later it becomes exceedingly rapid and feeble. Frequently cases in which ulcerative endocarditis develops are apparently of spontaneous origin. (For a discussion of this grave condition the reader is referred to the description of endocarditis in the section on Diseases of the Heart.) Among the blood-appearances during life are leukocytosis and a rather marked reduction in the red corpuscles, with moderate poikilocytosis. The blood-plaques are increased.

(f) **Cutaneous Symptoms.**—The most prominent is a mild yet decided grade of jaundice, that is probably hepatogenous in nature. Sweating has already been alluded to as a troublesome symptom, between the febrile paroxysms as well as immediately after. The skin finally shrinks from emaciation. Skin-eruptions are common, and particularly in the form of erythema, purpura, and pustules, and the general surface is often decidedly *hyperesthetic*.

(g) **Genito-urinary Symptoms.**—The urine is concentrated and urates are copiously deposited. There is albuminuria which may be due to the pathologic changes or may be, to some extent at least, ascribable to the febrile movement. The microscope discloses the presence of tubecasts, micrococci, pus- and (more rarely) blood-corpuscles. Albumose has been found in the urine.

(h) **Nervous Symptoms.**—The mind generally remains unclouded until an advanced stage is reached; then delirium sets in, and is followed by a terminal coma. This order of clinical events is not observed when metastatic purulent meningitis exists, the symptoms of which (hemi-

plegia, strabismus, ptosis, deafness, etc.) may appear at any period of the disease.

(i) Symptoms may be presented by the **joints and bones**. Metastatic arthritis, usually suppurative, is a not unusual concomitant, and in some cases it is combined with similar involvement of the long bones. Indeed, an acute osteomyelitis may be the only ascertainable source of the pyemia.

**Differential Diagnosis.**—The disease is often confounded with malarial intermittent fever, the distinctive features of which have been given under the differential diagnosis of the latter disease, but a diagnosis may always be made from the effect of quinin upon the fever. A few points of contrast, by means of which *septicemia* and *pyemia* may be differentiated, are tabulated below:

PYEMIA.	SEPTICEMIA.
Always associated with suppuration.	Suppuration may be absent, but there may be a sloughing wound.
Multiple chills.	A single chill.
Irregularly intermittent fever-curve.	Continued type of curve.
Profuse sweats accompanying febrile attacks.	Absent.
Rapid emaciation and profound prostration.	Less marked.
Nervous symptoms usually come on late.	Earlier.
Hyperesthesia.	Absent.
Slight jaundice.	Less marked (very faint).
Metastatic abscesses.	Absent.

**Prognosis.**—Pyemia may kill after an illness lasting but a few days. On the other hand, it may become more or less protracted, so that a chronic form has been distinguished. In this variety the symptoms are milder in character, and the tendency to the formation of metastatic abscesses is not as great in the acute form. Hence, while the prognosis is on the whole very bad, not a few of the more chronic cases terminate in recovery.

**Treatment.**—So far as the physician's province extends, the treatment is identical with that of septicemia. His efforts must be directed toward assiduously maintaining the vital powers. For the sweating the best agents are aromatic sulphuric acid and atropin; the latter may be given with agaricin (atropin, gr.  $\frac{1}{120}$ —0.0005; agaricin, gr.  $\frac{1}{8}$  to  $\frac{1}{4}$ —0.008 to 0.016), at bedtime. Prompt surgical interference must be resorted to, not only with a view to asepsis of the primary wound, but also to evacuating the primary and all secondary foci of suppuration.

## ACUTE ARTICULAR RHEUMATISM.

(*Rheumatic Fever.*)

**Definition.**—An acute febrile disease, the exact nature of which is unknown, though it is probably infectious. The chief local manifestation is a multiple arthritis, and its chief complications are cardiac (endo- and pericarditis). Hueter first advanced the germ-theory to account for



the disease, and, although the specific causal agent has not as yet been discovered, this view is the only one that offers a satisfactory explanation for the production of the lesions, the acute onset, the clinical course, and the complications of the disease. Rheumatic fever is subject to, and apparently obeys, the laws of infectious maladies in general, and the frequent involvement of the joints in many diseases belonging to this class may properly be regarded as supporting this theory. The disease is, in numerous localities, endemic, and at times also epidemic; but, on the other hand, in not a few regions (especially European) is practically unknown—*e. g.* England, Belgium, and Russia. Strümpell points out the fact that in Leipsic, where articular rheumatism is one of the most frequent of acute diseases, it has been observed for years that at certain times there are only a few cases, while at others there is a striking increase in the number.

**Pathology.**—The disease does not show peculiar lesions, and, although the joints are the chief seats of invasion, still in many instances, and even in aggravated cases, the changes presented are slight or altogether wanting. Usually the synovial membranes of the affected joints are injected and swollen, and their surfaces may be more or less coated with fibrin. The effusion is mainly serous, but contains fibrin and often leukocytes, and occupies the joints. A similar exudate infiltrates the periarticular tissues. The tendinous sheaths may also be inflamed; the cartilages in protracted cases may become eroded; and rarely a purulent exudate may be seen.

Fatal cases, except when death is due to hyperpyrexia, usually show the changes peculiar to endocarditis, pericarditis, or myocarditis, and less frequently those of pneumonia or pleurisy. The fibrin-factors of the blood are augmented.

**Etiology.**—**Bacteriology.**—Maragliano<sup>1</sup> has found in the blood of typical cases of acute articular rheumatism two micro-organisms—one resembling a bacillus and non-pathogenic, while the other is a micrococcus and (he thinks) the special infective agent of the disease. This organism resembles the staphylococcus aureus, but it is only half its diameter ( $0.5\mu$ ), and is massed in groups of six to ten. It is motile, is stained easily by anilin dyes, and is readily cultivated on gelatin. It develops at the usual temperatures, and especially at or about 98° F. (36.4° C.). Upon injection into rabbits the symptoms of acute articular rheumatism (polyarthritides, endocarditis, pericarditis, etc.) were reproduced. Guttman, Collin, and Sahli have found the staphylococcus in the articular exudate of patients suffering from complicated or recurrent cases of acute articular rheumatism, and Sahli is inclined to include the disease in the group caused by this organism. Netter, however, has found the streptococcus, and Lang a peculiar bacillus. Singer has noticed a relation between the number of pathogenic bacteria in the urine and the severity of the symptoms in cases of acute rheumatism; but Chvostek has not been able to confirm this result. A number of cases have been reported in which there is some evidence of direct contagion.

**Predisposing Causes.**—(1) An *infective lesion* (septic wound, attacks of angina, etc.) that has preceded for some time the appearance of the pain and articular manifestations may often be found, and this may be

<sup>1</sup> *Gaz. degli Ospedale e delle Clin.*, June 20, 1896.

conceived to form a portal of entry for micro-organisms (Sacaze). The frequency with which an attack of tonsillitis precedes the development of acute articular rheumatism almost indicates a pathological relation between the two diseases (Cheadle, Wade, Gerhardt). (2) *Seasons*.—The months of February, March, and April furnish the largest percentage of cases, though the disease is also quite prevalent in the remaining cold months; on the other hand, the disease may sometimes be especially frequent in summer. (3) “Catching cold” was formerly classed among exciting causes, but while this affection often follows exposure to abrupt changes of temperature, it merely predisposes to the disease. (4) *Climate*.—The disease is most prevalent in temperate latitudes, being rare both in the cold and tropical zones. (5) *Occupation* is of primary importance, especially if it entail oft-repeated or prolonged exposure to the influence of wet and cold or to severe changes of temperature. Hence those who follow certain avocations are attacked with great relative frequency—*e. g.* coachmen, laborers, sailors, and servant-girls. (6) *Age*.—Primary attacks are most common from fifteen to thirty-five years of age. Out of 655 cases, 80 per cent. occurred between the twentieth and fortieth years (Whipham). Cases are also rather numerous between ten and fifteen years, and I have met with 4 under the former age. Sucklings rarely suffer. (7) *Sex*.—Acute articular rheumatism is somewhat more common in men than in women, and possibly owing to the fact that the former sex more often follows predisposing occupations. (8) *Hereditary influence* can be traced in many families, and if chorea, recurrent tonsillitis, and chronic heart-disease are accepted as evidences of the rheumatic diathesis, it unquestionably plays a very important rôle. (9) Conditions of ill health, particularly digestive and hepatic disturbances, seem to exert a slight though decisive effect. (10) *Chronic endocarditis* renders its victims very prone to attacks of acute articular rheumatism, and some contend that the two diseases are etiologically one and the same.

An attack of acute articular rheumatism is not protective in character, and rather renders the individual more susceptible than before. In this respect the disease resembles certain other infectious diseases (pneumonia, erysipelas, etc.).

**Clinical History.**—Of the *incubation* period nothing is known, though prodromata, both local and general, may be observed. These may be malaise, slight fever, angina, laryngitis, etc., and last from a few hours to a day or two. The *invasion* is usually abrupt, with fever and synovitis, affecting one or oftener several joints, and a chill or a series of chilly sensations may accompany or precede the rise of temperature. The involved joints are tender, often red and swollen, and exhibit the local signs of a rapidly developed inflammation. Pain is a most prominent symptom. The medium-sized or larger joints (knee, ankle, and wrist) are first involved, and especially those of the inferior extremities; next the shoulder-, elbow-, and hip-joints; and lastly the fingers, toes, and intervertebral articulations. Quite unusual articulations may become implicated (*vide infra*). One of the chief peculiarities of the disease is in the fact that the joints that are affected are not all the seat of anatomic changes simultaneously, but that the process migrates from one joint to another from day to day, and often crosses from one side of the



body to the other. Sometimes this occurs at longer intervals. Hence the number of joints involved at one and the same time may be either few or many.

In cases of average severity the general features are subordinate to the local symptoms. The fever is usually moderate, the temperature not exceeding  $103^{\circ}$  F. ( $39.4^{\circ}$  C.), and the temperature-curve is of the irregularly remittent type, corresponding in severity with the joint-symptoms. Defervescence is by lysis. The skin is bathed in a copious perspiration which is not dependent upon a previous fall of temperature. Nervous symptoms are rarely observed.

The general course of the disease exhibits wide variations, both as to the duration and intensity of the symptoms. It may not outlast several days, appearing with mild symptoms; on the other hand, cases sometimes persist for six to eight weeks. The latter instances, and even typical cases, are apt to show brief periods of marked improvement, alternating with equally marked exacerbations of somewhat longer duration. Cases in which the symptoms are distinct from the start may terminate in recovery within a shorter time than those in which the features are of mild character. As will be seen hereafter, the disease frequently manifests complications, especially cardiac.

**Leading Symptoms and Complications in Detail.**—(1) **Joints and Surrounding Structures.**—As I have stated, pain is much complained of, and is greatly augmented by motion and by pressure of any sort. It may be out of all proportion to the degree of the anatomic changes. The joints affected are generally swollen (most markedly in the knees), and the swelling is due partly to effusion into the joint and partly to inflammatory edema of the periarticular structures. The sheaths of the tendons, the bursæ, and often the adjacent muscles and fasciæ exhibit inflammatory changes; hence it is usual to see an extension of the swelling for a variable distance from the joint, the backs of the hands often showing this to a marked extent. The skin may present a pink or rose-colored blush, often limited to circumscribed areas or taking the form of streaks.

In even mild cases there are usually two, three, or more joints involved, though it often happens that one bears the brunt of the disease, little complaint being made of others less severely implicated. Hence it should be a golden rule to examine carefully all the joints at each visit. Involvement of a single articulation (*monarticular rheumatism*) does sometimes occur, but the diagnosis of these cases offers great difficulties. On the other hand, an existing polyarticular rheumatism may become centered in a single joint and there linger with great obstinacy.

In severe cases numerous joints may be invaded, with an involvement of the joints of the symphyses, of the jaw, of the ribs, and the sterno-clavicular articulations. Under these circumstances the patient assumes a dorsal decubitus, and seeks to relieve his excruciating pain by holding his limbs in a semiflexed position and absolutely motionless. If now an attempt be made to change his posture, he complains pitiously of darting pains in the affected joints. The *fugacity* of rheumatic arthritis has already been alluded to.

The inflammation, however intense, may quickly subside in one joint, while at the same time an acute disturbance appears in another. Us-



ally resolution is complete, no trace being left of former inflammation, though the disease may recur in the joints primarily involved. Suppurative arthritis may supervene, though rarely, and its occurrence points indisputably to mixed infection. This complication may lead to ankylosis—a sequela which does not belong to pure rheumatism.

(2) **The Cardio-vascular Symptoms.**—The pulse is quickened to 100 beats per minute or over, but is soft and full, and when cardiac or other complications arise it shows special characteristics which are described in appropriate sections of this work. In rare instances it is very rapid, feeble, and irregular, apart from the influence of the cardiac involvement. The results of a careful blood-count show a high grade of symptomatic anemia, which may develop with marvellous suddenness. Leukocytosis is also present.

Great importance attaches to the cardiac affections that so frequently complicate this disease. They may arise in any case, even the mildest, or at any stage of the disease, and hence the conscientious physician cannot afford to neglect the matter of closely and regularly examining the heart. It must be recollected that the symptoms announcing the development of cardiac disease are neither constant nor characteristic. At first we may note an increase in the febrile movement, more or less palpitation, sometimes dyspnea, and precordial pains, which often do not amount to more than a sense of soreness. There may also be attacks of angina pectoris of apparently purely nervous origin (Strümpell).

(a) The most frequent cardiac complication is *acute endocarditis*, which is present in 25 to 30 per cent. of the cases. We are, however, sadly in need of reliable statistics upon this point. It usually takes the form of simple (verrucose) endocarditis, and affects most frequently the mitral valves. But, though usually indicated by an apical systolic murmur, it is hard indeed to eliminate the functional murmurs that may also develop in the course of this disease. Unless combined with the symptoms detailed above, the presence of a blowing systolic murmur does not afford trustworthy evidence of the existence of acute endocarditis. While it rarely endangers life and may leave no trace in the majority of instances the acute endocarditis does not undergo complete resolution, but leads to sclerotic changes and terminates in incurable chronic valvular disease.

(b) Next in the order of frequency is *pericarditis*, which may or may not be combined with the former. Its nature may be sero-fibrinous or plastic (less frequently), and in children the exudate is sometimes purulent; it is distinguished chiefly by its pathognomonic friction-sound, though also by other characteristic signs (*vide* Pericarditis). It is of graver import than endocarditis, so far as immediate danger to life is concerned, though it rarely proves fatal. I have witnessed two instances in which endocarditis preceded the arthritic manifestations, and the same observation has been made by others with reference to this complication as well as to pericarditis.

(c) *Myocarditis* is often present to a slight extent in rheumatic endocarditis and pericarditis when these occur independently of each other, but more often and to a more marked degree when endo-pericarditis exists. Hence it is far less common than either endocarditis or pericarditis. The changes and symptoms occasioned will be discussed under

Myocarditis. In this connection it should be pointed out that the condition weakens the cardiac walls and leads to dilatation of the ventricles (usually the left).

If we consider rheumatism an infectious malady, we can readily understand why the local manifestations should appear not only at the different articulations, but also in the cardiac structures, and, as we shall see, in other viscera.

(3) **The Skin.**—Rheumatism produces copious perspiration. The sweat emits a sour odor and gives at first an acid reaction, though later it may be neutral, and rarely alkaline. The temperature-curve in most cases is not materially influenced by the sweats. Occasionally the drops in temperature and the free sweats are concurrent, but the latter symptom is apt to persist despite the oscillations in the temperature. Sudamina appear, often in extensive crops. Among other skin-eruptions less frequently observed are forms of erythema (especially *E. nodosum*) and urticaria, which latter may be associated with purpura (*urticaria hæmorrhagica*). The association of the latter condition with polyarthritides is known as *peliosis rheumatica*, though, according to some writers, this is not rheumatic in nature. Cutaneous ecchymoses, and even extensive hemorrhages into the skin and from the mucous membranes—a general hemorrhagic diathesis—may also be encountered.

*Subcutaneous Rheumatic Nodules.*—In 1881, Barlow and Warner called attention to the fact that during and after acute articular rheumatism, particularly in children and young adults, small subcutaneous nodosities attached to the tendons and fasciæ may in exceptional instances be observed. These nodules are rather firm, somewhat movable, and usually painless. The skin over them is simply elevated, with no traces of inflammatory action. They are most frequently found at certain points of election (fingers, wrists, edge of the patella, malleoli, and over the back of the elbow), though also seen less frequently elsewhere; they may disappear, and after a brief interval reappear. On microscopic examination it is seen that round and spindle-shaped cells enter into their composition. I met with one case of the sort which occurred in a male aged forty-two years, in which acute articular rheumatism was also complicated with endo-pericarditis and pneumonia. Most of the nodosities were of the size of a bitter almond, a few being even larger, and the crop was extensive. The case proved fatal.

(4) **The Fever.**—The fact that the fever fluctuates materially in this affection has already been noted. It remains to be pointed out that if suppuration occur as a complication, the fever may be of the hectic variety; also that rarely hyperpyrexia is suddenly developed, and with it marked cerebral symptoms (restlessness, delirium, and sometimes convulsions, finally merging into stupor) are usually, though not necessarily, associated. This serious condition usually develops after several days of illness. Delirium may be present from the time of onset, though more often it comes on either shortly before or after the acute development of the hyperpyrexia. The pulse becomes excessively rapid and feeble and physical prostration extreme. The temperature may rise rapidly with slight interruptions until it touches 108° or 109° F. (42.7° C.), and as the fever reaches its maximum death usually ensues. The temperature may continue to rise after death. The cause of "hyper-



pyretic rheumatism" is not definitely known. It has been claimed that the intemperate are most apt to be attacked, but this belief is not corroborated by many clinicians. In a case of my own, however, of acute articular rheumatism, in which pericarditis with hyperpyrexia occurred, the patient was an "alcoholic." It is reasonably certain that the symptoms are due to an intense infection, with concentration of the poison upon the nerve-, and especially upon the thermal, centers.

(5) **The Muscular and Nervous Symptoms.**—It has been stated that the adjacent muscles and fasciæ may exhibit inflammatory changes. They may also show more or less swelling, and are often very tender to the touch, while in long-continued cases muscular atrophy ensues. The cause of this change is not clear, but the most likely view is that it results not so much from disuse of the muscles (the old theory), as from some trophic disturbance due either to the arthritis, or peripheral neuritis, or, to some extent at least, from extension of the rheumatic inflammation from the nearest articulation. Other theories have been advanced, but are scarcely tenable.

Mention has been made of the grave nervous symptoms that are attendant upon hyperpyrexia, but, independently of the latter condition, nervous phenomena may be present. There may be restlessness, sleeplessness, and active delirium, the latter being usually associated with a temperature of 104° F. (40° C.) or higher. In adynamic types, which are rare, low muttering delirium merging into stupor, and even coma, may be observed. Active mental symptoms are sometimes due to cerebral embolism secondary to acute endocarditis. When pericarditis is a complication, wild delirium, with or without hyperpyrexia, or the low muttering variety with stupor, is not unusual. The drunkard may develop delirium tremens. Coma, leading quickly to a fatal result, may develop without other previous or associated nervous symptoms, and DaCosta has reported cases in which a fatal coma was of renal origin, and hence uremic. Rarely coma develops during the period of convalescence. *Convulsions* may be noted, generally preceding the coma, though rarely as an independent symptom. *Melancholia* may arise in the course of the disease, but more frequently at its close. *Meningitis* must be numbered among the rarest of complications.

*Chorea* is a not infrequent sequel of this disease in children, and more rarely is associated with it. Of 554 cases analyzed by Osler, in only 88 were chorea and rheumatism associated. These instances may or may not be accompanied by acute endocarditis.

(6) **Pulmonary Symptoms.**—Pleurisy occurs, and is generally excited by an extension of inflammation from the pericardium, and from the pleura the inflammatory process may be propagated through the diaphragm to the peritoneum. Bronchitis is sometimes present, but is rarely a part of the rheumatic morbid process; it is secondary, and in most instances is occasioned by the co-operation of the factors that are at work in every disease in which enforced recumbency and great prostration coexist. In like manner, broncho-pneumonia may be produced. Lobar pneumonia rarely occurs, and is confined to aggravated cases, but pulmonary congestion is occasionally seen, and may prove fatal. Pulmonary complications are also prone to develop secondarily to pericarditis, and especially to endo-pericarditis.



(7) **The Renal Symptoms.**—The urine is diminished in amount, is high-colored, and of high acidity and density. The standing specimen deposits urates. As in other infectious diseases, there is commonly present a slight febrile albuminuria, but acute nephritis is extremely rare. The chlorids are sometimes diminished, but rarely absent.

(8) **The spleen** is slightly enlarged in some cases. The saliva has sometimes an acid reaction, and, according to certain writers, the sulfo-cyanids are in excess.

**Clinical Peculiarities of Acute Articular Rheumatism in Children.**—The arthritic symptoms in children are in abeyance while endocarditis and pericarditis are predominant, and these cardiac conditions may appear before the joint-lesions are observed, but it is quite probable that endocarditis follows the joint-lesions twice as frequently in children as in adults. Parsons lays stress upon reduplication of the cardiac second sound, audible at the apex only, as an indication of the development of endocarditis. This sign is to be distinguished from reduplication heard at the base, sometimes as the result of Bright's disease and sometimes as the consequence of pulmonary obstruction. Acid sweats are slight in children. Rheumatic tonsillitis is quite common, and may precede, accompany, or follow attacks of rheumatism in children. Erythema is a frequent concomitant, and is often mistaken for scarlatina. The febrile movement lasts usually but a few days.

**Diagnosis.**—The acute development as a primary affection of poly-arthritis with fever is a combination of symptoms on which a diagnosis can be usually based with considerable reliance. Pyemia must be carefully separated, since here also we have the implication of the joints with fever. In pyemia, however, the general condition is more grave as a rule; fever is more apt to precede the local manifestations, and the curve is irregularly intermittent, whereas in rheumatism it is irregularly remittent. Rigors also occur in pyemia at irregular intervals, accompanied by a rapid rise of temperature, and are absent in rheumatism; suppurative processes are early set up in the various viscera and skin, which latter also shows slight but decided jaundice. The symptoms, both general and local, in acute articular rheumatism fluctuate greatly, while this is not so in pyemia.

The multiple swelling of the joints which develops after labor is to be regarded as septic in nature. *Gout* will be distinguished from rheumatism in connection with the consideration of the former disease (*vide* p. 398).

*Monarticular rheumatism* is with difficulty differentiated from a group of affections which simulate it closely. (1) The so-called *gonorrheal rheumatism* often affects a single joint, especially the knee; but in this disease there is usually a definite history of recent infection, and the local features (pain, swelling, etc.), unlike true rheumatism, are far more pronounced than the general. The course of gonorrheal arthritis is longer in duration, and is generally connected only with a single joint from the start; while acute articular rheumatism almost always begins as a polyarthritis, with subsequent fixation in one articulation. Cardiac complications are rare in the former disease.

(2) *Acute osteomyelitis* is generally single, and is sometimes mistaken for rheumatism, from which it differs, however, in the localization of the

lesions in a single joint from the start, the greater prominence of the local symptoms, and in the implication of the epiphyses and the shaft of the affected bone rather than the joint, and in the graver general symptoms from the time of onset.

(3) There is a liability to mistake the acute arthritis of infants for rheumatism. This attacks by preference the hip or knee, and is a purulent inflammation due to pyemia (Townsend), hence having no relation to the disease under consideration.

(4) *Scrofulous arthritis*, particularly in children, has been confounded with rheumatic monarthritis. The former is less indurating, the swelling presented is less symmetric, and the course is far less acute than that of the latter.

(5) In the course of the hemorrhagic diseases, scurvy, purpura, and hemophilia, effusion into the joints, either hemorrhagic or serous in nature, occurs with great frequency and is associated with rheumatic pains. The differential diagnosis is to be made from the tendency to hemorrhage, and in scurvy by the lesions of the gums. The absence of fever is usually decisive: unfortunately, it is frequently present in these joint-affections.

**Prognosis.**—Recovery is the general rule. As in other infectious diseases, so in rheumatism, the chief immediate danger springs from the great intensity of the type of infection, as manifested in hyperpyrexia with grave nervous symptoms, the development of the general hemorrhagic diathesis, etc.—happily rare occurrences in this disease. Certain complications, such as pericarditis, endo-pericarditis, pneumonia, etc., may render rheumatism grave or even hopeless, and rarely the endocarditis that complicates the disease is of the ulcerative variety and leads to fatal pyemia. Pulmonary embolism may occur during the course of acute articular rheumatism, causing speedy death.

The influence of personal factors may impede recovery, such as intemperate habits, great obesity, the existence of previous organic disease of the heart or Bright's disease, etc.

**Treatment.**—(1) **Sanitary Environment, Diet, and Stimulants.**—The sick-room should be well ventilated, and its temperature maintained at 65° to 70° F. (18.3°–21.1° C.), but draughts should be avoided. The patient should be lightly dressed in flannels and covered with a sheet of the same material. The *diet* should be liquid and nourishing, milk being the best food-article as a rule, and being well borne. Skimmed milk, milk and Seltzer water, buttermilk, milk and lime-water, meat-juice, egg-white, and solids (other than animal) may all be employed if ordinary milk cannot be taken in adequate amount. I begin the use of the more nutritious and easily digested forms of animal food as soon as defervescence has occurred. *Stimulants* may be employed if indications for their use are present, and the customary mode of administration may be followed. Fortunately, these do not arise as often as in many other affections belonging to the class.

(2) **Internal Therapeutics.**—There has been of late a surprising unanimity among clinicians in commending the use of the salicylates in the treatment of this disease—more so than at any previous time since their introduction. They are employed in most of the larger hospitals both in Europe and America. Differences, however,



relating to the mode of administration and the particular salt to be selected still exist. Wood<sup>1</sup> favors ammonium salicylate, for the reasons that it is freely soluble, is rapidly absorbed, and when given in sufficient amount quickly produces the symptoms that mark salicylic action, while, in addition, it is less depressing than the other salts of salicylic acid. It is best given in milk and is usually well borne. My experience with this salt in acute articular rheumatism, though as yet somewhat limited, has been satisfactory. Until the present time sodium salicylate has met with more general favor than any other single salt of salicylic acid. The pure acid is also used, though not to any great extent at the present day. As regards the mode of administration, the total daily amount taken is of higher importance than the size and frequency of the dose. The amount given in twenty-four hours should not exceed 2 drams (8.0), while often  $1\frac{1}{2}$  drams (6.0) of the sodium or ammonium salicylate is sufficient. My method is to give gr. x (0.648) every two hours during the first day, or until the pain and other local features have largely disappeared; then the remedy is given at longer intervals, but not omitted entirely. In this manner fresh exacerbations are most probably averted. If the latter occur, however, larger doses must be instituted, so as to cut them short. Some recommend that the medicine be stopped as soon as the pain has been controlled. If salicylic acid be employed, it should be given in capsules. According to certain observers, salol is to be preferred to either the pure acid or the salicylates: in my experience, however, the use of this drug has not been followed by good results in the severe acute forms of the disease. Doubtless the reason for this lies in the fact that salicylic acid can neither be introduced into the system in sufficient amount nor rapidly enough in the form of salol.

Kinnicut has recommended the employment of the oil of winter-green, a salicylic compound which does not generally produce the unpleasant toxic symptoms so apt to be excited by the salicylates or salicylic acid. The dose is  $\mathfrak{M}$ x-xx (0.60-1.25), given in capsules or in milk, to be repeated every two hours. Salicin (gr. x-0.648, every hour, increased to gr. xv-0.972) is sometimes efficacious and invariably agrees. Salophen, in daily doses of 1 dram (gr. xv-0.972, every four hours) until pain is relieved and temperature reduced, has been warmly advocated. Though almost specific in its effects, the drug does not prevent either the spread of the disease to new joints, fresh exacerbations, or cardiac complications. Sodium salicylate enemata (3j-4.0—of the salicylate and  $\mathfrak{M}$ x-0.60—of the tincture of opium in each injection) may be of advantage in certain cases. The remedy is absorbed from the rectal mucosa, though more slowly than from the stomach.

The treatment with the salicylates or salicylic acid mitigates the fever, relieves the pain, and shortens the stay in bed by a few days, but does not curtail convalescence. The statistics of Williams go to show that the salicylate treatment also tends to protect against the development of cardiac complications, though it does not seem to influence the course of the complications once they are established. In my experience the alkaline treatment operates potently to obviate the occurrence of the heart-complications and shortens the period of convalescence, but exerts slight, if any, influence upon the fever-curve and pain. These

<sup>1</sup> *University Medical Magazine*, Jan., 1895.



facts led me long since to use the specific and alkaline treatment in combination, giving, in addition to salicylates or other salicylic compounds, as above indicated, an alkaline remedy, such as sodium bicarbonate, potassium citrate, etc., in sufficient doses to render, and then maintain, the urine of slightly alkaline reaction.

There are a few other remedies that should be referred to, and, although more or less serviceable, they are without specific influence. The foremost among these is antipyrin, which may be used advantageously during the acute period of the disease, the heart being at the same time watched over carefully. Loomis says of the drug: "I have come to employ it almost to the exclusion of all other remedies for the relief of the arthritic pain, and in many cases it has seemed markedly to shorten the duration of the disease." Potassium iodid and the preparations of colchicum also belong to this category, and should be tried. Their effects are most beneficial in cases that drag on after the acute stage is over. Recently lactophenin has been brought forward by Roth as a most useful remedy in acute rheumatism, but it does not rival the salicylates.

(3) **Local Measures.**—These occupy a subordinate place in the management of acute articular rheumatism. Their number is legion, but only a few of the more valuable can be adduced here. In mild cases the affected joints should be wrapped in cotton batting or in flannel. If the pain is severe despite the use of the salicylates internally, fomentations as hot as can be borne or hot cloths lightly wrung out of Fuller's lotion (sodium carbonate,  $\text{ʒvj}$ —24.0; laudanum,  $\text{ʒj}$ —30.0; glycerin,  $\text{ʒij}$ —60.0; and water,  $\text{ʒix}$ —270.0) are beneficial. As salicylic acid is absorbed through the skin, it has been combined with other agents for local use in the following formula:

R <sub>y</sub> . Acid. salicyl.,	
Lanolini,	$\bar{a}\bar{a}$ . $\text{ʒij}$ (11.65);
Ol. terebinthinæ,	$\text{ʒij}$ (11.25);
Adipis,	$\text{ʒij}$ (11.65).

M. et ft. ung.

Sig. Rub freely over the affected joints and follow by wrapping in cotton.

Cold compresses and the ice-bag to the joints have been strongly advised, particularly by German authors. The affected joints should be kept at perfect rest, and this is best accomplished by padded splints and a roller bandage. Blisters near to the joints involved and the light application of the Paquelin thermo-cautery are sometimes serviceable, but they are to be thought of only when the above-mentioned local means have failed.

The treatment of the *complications* will be considered under their appropriate headings. I desire, however, to emphasize the fact in this connection that should hyperpyrexia occur during the progress of the affection, it is to be relieved by cold baths, since large doses of antipyrin or other internal antipyretics are of themselves dangerous. It may also be stated that the cardiac complications—endocarditis, pericarditis, and endo-pericarditis—rarely require special remedies. If marked cardiac asthenia appears, as indicated by the feeble first sound, the salicylates

may be replaced by salicin, which is less depressing in its effect upon the heart. Cardiac stimulants may be required. A copious pericardial effusion calls for paracentesis (*vide* Sero-fibrinous Pericarditis).

During convalescence the patient should not be allowed to get out of bed too early. My own rule has been to keep him in bed for a week after the temperature has returned to the normal and after the pain has disappeared, except it be during the hot season. These precautions are taken to avoid the occurrence of relapses. After the patient goes into the open air he should be told to avoid cold, and wet in particular. During this period iron is to be employed until the blood-examination fails to show anything abnormal. For the stiffness and swelling that sometimes persist, or disappear very slowly after the acute attack, massage and the application of hot water or warm baths seem to yield the best results.

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### SUBACUTE ARTICULAR RHEUMATISM.

THIS is, as a rule, a sequela of acute rheumatism, and may occur, though rarely, in persons who have not had a previous acute attack. Both the local and general features are of a mild type, but the course is apt to be prolonged into two, three, or more months. Usually the local symptoms are confined to one or two of the larger joints, with little swelling or redness, and the pain is slight except on movement. The temperature rarely exceeds 101° F. (38.3° C.), and at times may be practically normal. Though the course is prolonged, the joints usually return to their normal state; occasionally, however, the disease becomes chronic. As in the acute form, so in the subacute, anemia becomes well marked and cardiac complications are not uncommon, particularly when the disease occurs in children.

The **treatment** embraces, in addition to the usual antirheumatics, the use of iron, quinin, cod-liver oil, and, when practicable, a change to a warm climate. The affected joints demand hot applications and massage.

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### GONORRHEAL ARTHRITIS.

**Definition.**—A septic synovitis caused by the gonococcus, and hence having no connection with true rheumatism. It usually manifests itself toward the close of an attack of gonorrhea, but it may develop during the active stage of the disease or at any period during the course of gleet.

**Pathology.**—The signs of ordinary synovitis are generally found in the affected joints, though, not rarely, the inflammatory process is periarticular rather than articular. Under these circumstances the inflammation may travel along the sheaths of the tendons for a considerable distance. Synovial effusion may occur, and rarely may be purulent, this being most frequent in gonorrheal inflammations affecting

the wrist and hand. Gonococci have been found in the effusion, though this is not generally the case, and it is now thought by many writers (Finger, Councilman, and others) that the gonococcus may be the only infective agent concerned in the morbid process. Others contend that the metastatic inflammation of the joints is due to the presence of pyogenic cocci, since they have been found to be frequent companions of the gonococcus. In this and other forms of secondary inflammation it must not be forgotten, however, that gonorrheal arthritis may be due in great part to the absorption of ptomaines from the urethra. The disease occurs much more frequently in men than women, and some claim that it may follow any urethral discharge in the male or may be associated with menstruation or leukorrhea in the female.

**Clinical Symptoms.**—Two leading varieties, acute and chronic, are encountered. (1) *Acute Gonorrheal Arthritis*.—This may be very mild, amounting merely to slight fugitive pains and some stiffness of one or more joints, without noticeable swelling or redness. The typical, acute form, however, presents the symptoms of a severe fibrinous or sero-fibrinous inflammation of a single joint, developing quickly. The pain is often violent; there is swelling of the joint with extension along the course of the tendons, and the condition is obstinate. Unless pus be present (a rare event) the constitutional features do not correspond in severity with the local, there being little fever and slight impairment of the general health. There are many instances in which the complaint begins as a polyarthritis, with subsequent concentration upon one or two of the larger articulations, especially the knees or ankles. Fibrinous ankylosis usually remains as the resulting condition.

*Acute endocarditis* may be of gonorrheal origin. In the inflammatory products of this condition Hering has found the gonococci, as has also Councilman, in the heart-muscles (gonorrheal myocarditis). Rarely, gonorrheal endocarditis assumes the ulcerative or malignant form. As the result of invasion of the blood by the gonococci *suppurative arthritis* may also be occasioned, and now the clinical picture is that of general septico-pyemia. I observed one case in which *pleurisy* was associated, and among the numerous widespread complications *iritis* deserves special mention.

(2) *Chronic Gonorrheal Arthritis*.—This occurs (a) as a serous effusion (*hydrarthrosis*), and (b) as a chronic inflammation of the articular and periarticular structures (synovial membranes, bursæ, periosteum, and tendons with their sheaths). The former is usually monarticular, settling with especial frequency in the knees, and may be wholly painless. The latter is more or less painful—causes dense swelling of the joint, and frequently of the structures for some little distance above and below the latter. Both forms lead to great restriction of motion.

The **diagnosis** cannot be determined positively apart from the history of urethral infection. The acute form is distinguished chiefly from *acute articular rheumatism* by the intense pain, the extent to which the periarticular tissues are involved, and the negative character of the general symptoms. The chronic variety must be discriminated from *chronic synovitis* due to other causes, and this often proves a difficult task.

**Treatment.**—I have never seen the slightest benefit from internal



medication in gonorrheal arthritis, except possibly from the use of mercury until the patient was brought decidedly under its influence. *Local measures*, however, are of paramount importance. Absolute rest to the part is indicated, and the limb should be placed upon a splint; then, after making an appropriate anodyne application (ungt. ichthyol or ungt. belladonnæ), it should be bandaged as firmly as possible. Before doing this in acute cases the patient should be anesthetized, and after the procedure, if pain be great, a hypodermic injection of morphin may be given. In *chronic forms* the aim should be to remove the effusion (if present) and the swelling, and to restore the natural motility so far as possible. For the latter two indications massage and passive movements are best. Swelling may also be diminished by the use of the thermo-cautery at intervals, and blisters are highly serviceable in causing a disappearance of the effusion. Careful surgical attention should be bestowed upon the urethral or vaginal condition.

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## VARIOLA.

(*Small-pox.*)

**Definition.**—Variola is an acute contagious disease, characterized by its sudden onset and severe period of invasion, followed by a remission of the fever and an eruption of papules, which pass through the stages of vesicle, pustule, and scab. The stage of pustulation is accompanied by secondary fever.

**Historic Note.**—Small-pox has existed from the earliest antiquity in India, Africa, China, and other Eastern countries. During the thirteenth century (1241) it entered England, in the early part of the fourteenth Ireland, and in the latter part of the fifteenth Germany. In 1507 it was imported to America, and first appeared in the West Indies; a little later (1520) the Spanish troops conveyed the disease to Mexico, where it destroyed not less than three and a half millions of people in its pestilential march. It was brought to the United States from Europe in 1649, and gained its first foothold in Boston, whence it progressed at intervals in a westerly direction till it finally reached the western coast-line nearly a century after its first introduction.

At the present day there is a very limited opportunity to observe the affection except in its modified form (*varioloid*), since small-pox certainly does not prevail in an epidemic form where vaccination is practised with fidelity, and only among uncivilized peoples who are ignorant of or indifferent to this certain prophylactic power.

**Pathology.**—The eruption of small-pox consists in an inflammatory cellular infiltration of the *rete mucosum* and has four successive stages—(1) *Papular*, (2) *Vesicular*, (3) *Pustular*, and (4) *Scab*.

(1) *The Papule.*—At first there is a hyperemia of the papillæ of the skin appearing as small red spots. These soon become round, discrete patches that may be rolled like shot under the skin, and then, becoming elevated, owing to the increase in the cells in the *rete mucosum*, they form the typical *papule*.

(2) The *vesicle* appears at the apex of the papule, and results from a circumscribed elevation of the superficial layer of the epidermis in consequence of the mechanical pressure exerted by the fluid exudate, which is excited by active peripheral inflammation. The vesicle is not unicellular, but is loculated (by fibrinous reticuli), and contains serum, leukocytes, fibrin-filaments, etc. If a section of a vesicle in the very early stage be made through the deeper layers of the *rete mucosum*, an area of coagulation-necrosis is observed, which is due to the presence of micrococci (Weigert). The vesicle shows central umbilication, which corresponds with the necrotic area, and is well marked just before the pustules are formed.

(3) The *pustule* is formed by the filling of the reticuli with leukocytes. Cellular infiltration and swelling of the true skin beneath the pustule occurs, as a rule, as the result of diapedesis. Moreover, suppuration may involve the *cutis vera*, and as a consequence scarring results. When the suppuration is limited to the *rete mucosum* pitting does not take place. In hemorrhagic small-pox the reticuli are occupied by an abundance of red corpuscles which have passed in from the adjacent blood-vessels, and may infiltrate the upper as well as the deeper layers of the epidermis surrounding the vesicles or pustules. The pustules may dry up, but commonly rupture, and in either case the result is (4) *scabbing*.

The mucosa of the mouth, pharynx, and, rarely, the esophagus and the rectum may be the seat of a variolous eruption, and the plaques of Peyer may be somewhat swollen. The eruption also appears in the larynx, the trachea, and bronchi, where ulcers rather than true pustules are seen, and the conjunctiva and nasal mucosa frequently show the specific lesions.

Hemorrhagic small-pox presents extravasations occurring in the serous and mucous membranes, the connective tissue, the parenchyma of the various viscera, and also, though much less frequently, in the nerve-sheaths, bone-marrow, blood-vessel walls, and the muscles. In this form the spleen is firm (Ponfick, Osler), and the liver is sometimes enlarged and the subject of fatty degeneration. Hemorrhagic infarction of the lung occurred in 5 out of 7 cases examined by Osler. "In 4 instances the pelvis of the kidney were blocked with dark clots, which extended into the calyces and down the ureters," and in a proportionate number of cases Peyer's glands were swollen.

**Secondary Lesions.**—The catarrhal inflammation of the larynx may extend in depth till it touches the perichondrium of the cartilages (perichondritis), and a croupous exudate in the larynx may often coexist with edema. Lesions are present in the lungs, some of them frequently (general bronchitis, broncho-pneumonia), and others rarely (hypostatic congestion, lobar pneumonia), and a low type of inflammation of the *pleura* may be observed. Cloudy swelling, diffuse inflammation, and sometimes fatty degeneration of the *liver*, have been noted, the *spleen* being enlarged and pulpy as a rule. The *heart* may show myocardial alterations—chiefly parenchymatous and fatty—and rarely endocarditis and pericarditis occur in the nature of true complications. The *kidneys* show cloudy swelling, and occasionally nephritis develops, although not until quite late. Attention should be called to the observation of Wei-

gert, who found that at the commencement of the stage of suppuration the microscope revealed "small-pox cylindric masses" in the various organs (liver, spleen, kidneys, and lymphatic glands). The cylindric masses are in reality capillaries filled with micrococci, which are derived from the eruption, and probably find their way into the circulation through the lymphatics. These small areas of coagulation-necrosis may be the seat of leukocytic infiltration, which in turn may result in a circumscribed miliary abscess if septic material be also absorbed from the pustules. No special microscopic appearances are presented by the blood.

**Etiology.**—**Bacteriology.**—Loeb and Pfeiffer<sup>1</sup> have described certain protozoa found in the blood or in local lesions, to which they attribute an etiologic significance. J. Christian Bay<sup>2</sup> has isolated from small-pox lymph and vaccine points an organism (*dispora variolæ*) which he believes to be the *causa morbi* of small-pox and vaccinia. The long diameter of the bacterium measures  $0.6\mu$ – $1\mu$ , and the short diameter from  $0.2\mu$ – $0.3\mu$ . The organisms contained two spores, one at either end, and when reared on artificial media developed in colonies, and were readily stained with anilin blue or violet. A further study of the dispora, however, is essential, and experiments having for their object the reproduction of the affection in the lower animals have not been successfully conducted as yet. Hence the exact rôle played by the organism in the causation of variola has not as yet been definitely determined.

**Predisposing Causes.**—The *receptivity* for variola is wellnigh universal, though in rare instances persons are insusceptible, and among the few who have enjoyed complete immunity against small-pox were three distinguished physicians—Diemerbroeck, Boerhaave, and Morgagni. It may be said that one attack confers permanent immunity, but in rare exceptions a second or even a third may occur. *Vaccination*, also, if successful, affords future protection against variola, but to this rule exceptions are not infrequent.

**Age.**—All periods of life are liable to the disease, but the very young are affected in a relatively larger proportion than older persons. During the entire puerperal stage there is an increased liability to the disease. It rarely affects the fetus in utero, and most babes even, who are exposed to the virus at the time of birth, will not take the disease if immediately and successfully vaccinated.

**Sex** is without influence.

**Race.**—Among uncivilized peoples variola spreads with frightful rapidity, the negro and other very dark races being affected in larger numbers and more severely than whites. A dread of the infection predisposes to its occurrence.

**The Contagion; where Found; Modes of Conveyance and of Infection.**—One case of variola is *primâ facie* evidence of the existence of another, and that the poison from the latter was somehow transferred to the former. The specific poison exists in the blood and in the secretions and excretions (most probably), but mainly in the pustules and dry scabs and in exhalations from the lungs and skin. Its conveyance from the sick to the healthy or from one person to another is not a difficult

<sup>1</sup> Loeb, *Centralbl. für Bacteriologie und Parasitenkunde*, ii. 353, 1887; Pfeiffer, *ibid.*, ii. 126, 1887.

<sup>2</sup> *Medical News*, January 26, 1895, vol. lxvi. p. 94.



matter, and may take place in one or other of the several following ways: (a) *Inoculation* with either the blood or the contents of the eruption or the dissolved dry scabs is followed by variola. (b) Contact with, or proximity to, a patient suffering from small-pox is very apt to convey the poison, with resulting variola in the person thus exposed. To what distance the contagion can be conveyed through the air is not known, but it is probably considerable; and all authors are agreed that it is one of the most infective diseases with which we are acquainted. It is contagious from the earliest active stage to the end of convalescence, and, according to some observers, even during the stage of incubation. (c) Transmission by fomites is common, the poison adhering to clothes, body- or bed-linen, etc., and evidence is not wanting to show that the poison is highly tenacious of pathogenic power. Its vitality is retained after death, and the room occupied by a patient, the bedding, and the articles of furniture all serve to convey the disease unless thorough disinfection be enforced. The avenue of entrance for the poison into the system is not known, but it is most probably the respiratory tract, the poison being inhaled and thence taken into the general circulation.

**Clinical History.**—**Incubation.**—This stage varies with the mode of communication of the poison. If following inoculation, the symptoms appear in six or seven days; when originating in infection, usually in twelve days, though this stage may be either lengthened by a day or two or shortened to an equal extent. During a portion of this period complaint may be made of certain ill-defined symptoms, but these are usually absent, and the onset is sudden and accompanied by characteristic signs. These are—a *severe rigor, high fever, headache, and intense lumbar pains*. Instead of the usual severe rigor, repeated chills, extending over twelve to twenty-four hours, may occur, the headache, intense pain in the loins, and the fever continuing for several days unless relieved. During the same period the respirations are accelerated, the pulse becoming decidedly more rapid, and there may be general bronchitis. The tongue is coated and the patient may make complaint of slight pharyngitis. There is anorexia (often complete), generally vomiting, and constipation or rarely diarrhea. Restlessness, wakefulness, delirium, and stupor are the most important nervous symptoms observed. Infective albuminuria is not uncommon, and in the female menstruation is apt to be brought on.

The physical signs are few, and consist of a few dry and, later, moist râles, heard on auscultation. Palpation detects splenic enlargement. From the second day the so-called initial rashes may appear: (a) the diffuse scarlatinous eruption, which in no way differs from ordinary *scarlatina*; (b) the measly eruption, which may be diffuse and identical with that of true measles. Either associated with these or occurring independently there may be a hemorrhagic eruption (usually purpura), the petechiæ coming out by natural selection, mainly upon the hypogastric region or the inner surfaces of the thighs and in the axillæ (Simon). Rarely the knees, elbows, and extensor surfaces present this eruption. The initial rashes occur in a considerable proportion of cases (10–15 per cent.), and of these the petechiæ outlast the other pre-variolous eruptions. The stage of invasion just depicted lasts three days as a rule, and the intensity of the symptoms is generally in

direct proportion to that of the stages that follow. At the end of the third day or on the fourth the temperature declines rapidly, while at the same time the true variolous eruption appears upon the skin and mucous surfaces. Now begins the *stage of eruption*, which develops first upon the face, particularly upon the forehead and the hairy scalp, and spreads in a downward direction till it reaches the legs, where it last appears. The skin in the femoral triangle rarely shows the true variolous eruption. Each pock passes through the various stages noted in the pathologic description—viz. papule, vesicle, pustule, and scab; and when the stage of pustulation has been reached a secondary fever develops. During the following remission of fever the headache, lumbar pains, etc. subside. The fever of suppuration which then succeeds is accompanied once more by marked constitutional disturbances, particularly nervous derangements (wild delirium, etc.), and at this time complications are also apt to develop. On the eighth or ninth day of the eruption (the twelfth or thirteenth day of the disease) the pustules begin to dry up, forming yellow crusts; the redness and swelling of the skin subside; and two or three days later the scabs loosen and are thrown off. During this stage the fever again declines in company with the constitutional symptoms, and convalescence ensues. As previously stated, when suppuration involves the true skin scars are the inevitable result, and these remain to the end of life. The hair drops off sometimes, even to the extent of total alopecia, but is generally renewed.

**Leading Symptoms and Complications.**—(a) **Eruption.**—As before stated, the eruption makes its appearance at the end of the third or on the fourth day, coming out first upon the forehead, particularly along the border of the hairy scalp, and spreading in a downward direction in regular progression. It appears in the form of slightly elevated maculæ, which are at first of a pale-red color, and later assume a darker red hue, resembling small fleabites. These increase in size during the next forty-eight hours, at the end of which period they are developed into (1) *papules*. The change of character is accompanied by itching and burning of the skin-surface. To the feel they are papular—like shot under the skin—and if the finger-tips be rubbed over them lightly they are distinctly satin-like. The eruption is always most abundant upon the face and scalp, while the hands and fingers are the next most favored seats. At the end of the third day (the sixth day of the disease) the conical apices of the papules contain liquid, forming thus (2) *vesicles*. The latter increase in size till the entire papule is converted, at the same time acquiring more and more decidedly a central umbilication. Puncturing a vesicle does not cause it to collapse, but allows only a small portion of its liquid contents to escape, owing to its reticulated character. As the vesicle increases in size its contents become opaque, and in three days more, or about the sixth of the eruption, the vesicles become (3) *pustules*. Umbilication now disappears, and the pustule looks full and well rounded, and is surrounded by a red border or “halo.” If the pocks be close set, as on the face, wrists, and fingers, the intervening skin is inflamed and swollen and the itching and burning become almost intolerable. The pustules may coalesce along their edges, and thus the eruption becomes confluent. The eyes are closed as the result of swelling and tumefaction of the face, and the hands and feet assume a rounded,



ball-like appearance. The face, as a whole, is markedly misshapen and is ultimately disfigured. When the pus is not liberated (a comparatively rare event), its desiccation begins on the ninth day (the twelfth day of the affection); if the pustule is ruptured earlier (as when confluence occurs), it begins at an earlier day. (4) The *scabs* now form, and remain until about the twelfth day of the eruption, and when pits or scars result they are at first distinctly hyperemic, but gradually fade until at last they remain as permanent whitish spots.

The eruption upon the mucous membrane develops simultaneously with that of the skin, and among favorite surfaces for its appearance are (as pointed out under Etiology) the mouth, tongue, soft palate, and pharynx (causing dysphagia), the nasal chambers (causing coryza), the larynx (causing hoarseness), the trachea and bronchi (causing bronchitis). This mucous efflorescence does not proceed to the development of pustules, but forms ordinary ulcers as a consequence of early maceration of the superficial layers of the mucosa, and these ulcers also may become confluent.

The *skin* also presents certain complications that are always secondary and are deserving of mere mention (erysipelas, abscess, gangrene, bed-sores, etc.).

(b) **The Fever.**—The temperature at the onset rises rapidly, and may touch  $103^{\circ}$  or  $104^{\circ}$  F. ( $40^{\circ}$  C.) on the first day, its range being high and of the continued type during the invasion period. Evening temperatures of  $105^{\circ}$  F. ( $40.5^{\circ}$  C.) or higher may be observed, and in three days (or with the first appearance of the papules) the temperature remits, but does not intermit in true variola. It remains at a low elevation till the stage of suppuration is reached, when a fresh rise, sometimes to its

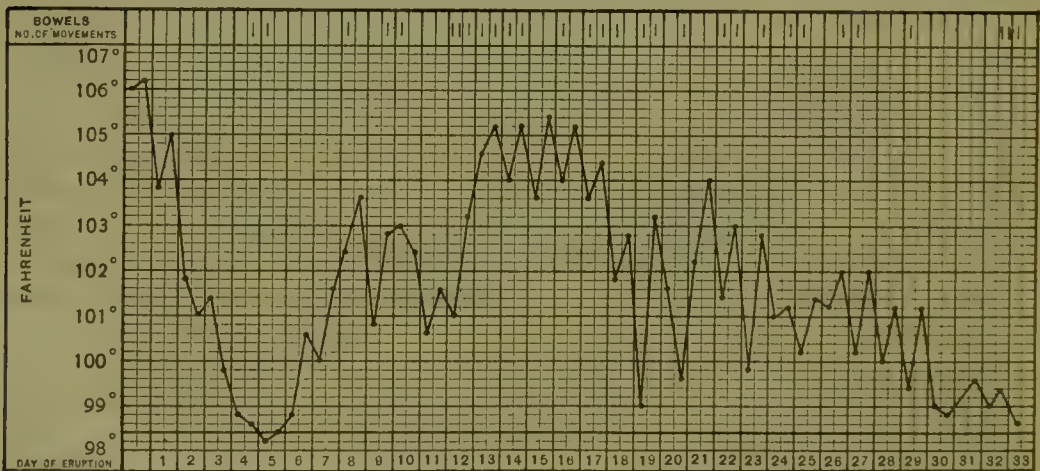


FIG. 21.—Temperature-chart of a case of variola, from a patient in the Municipal Hospital, Philadelphia. A. F.—, aged three years; not vaccinated.

original height, occurs. This secondary fever-curve is apt to show a decided irregularity, with exaggerated points of elevation and deep remissions. The latter may be due to complications, but are most generally the result of septic absorption (the fever of suppuration). This period lasts from one to three or four days in typical cases. When desiccation of the pustules begins defervescence also commences, and proceeds in a gradual manner by lysis. There may be a post-variola rise, and if so its presence is to be attributed to some sequel or other.



(c) **The Circulatory System.**—The pulse is soft and much accelerated (100 to 130) and of good volume during the invasion stage. It is slower during the period of remission, only to be greatly increased in frequency during the second stage of fever. During the latter period it may, owing to cardiac failure, become very rapid, much enfeebled, and finally irregular or even intermittent. The pulse-rate will vary according to differences in the previous general condition of the individual. Simple endocarditis rarely, and pericarditis somewhat more frequently, occur as complications.

(d) **Respiratory Tract.**—The *laryngitis* and *pharyngitis* which are due to the presence of pocks in the mucosa of the respiratory passages have already been mentioned. The most serious events, however, connected with the respiratory system originate in secondary infections, if we except *laryngeal perichondritis* with edema of the glottis, the latter perhaps being the result of a direct extension of the pock-ulcers to the perichondrium. Chief among the secondary complications is *broncho-pneumonia* (inhalation pneumonia), and *lobar pneumonia* also occurs, though rarely. *Pleurisy*, however, is not infrequent, particularly as an associated condition in broncho-pneumonia.

(e) **The Digestive System.**—A frequent seat of the variolous efflorescence is in the buccal and pharyngeal mucosæ, where it causes exceedingly unpleasant symptoms (*vide ante*). It may also be an agency in predisposing to a secondary inflammation in adjacent organs—*e. g.* suppurative otitis media, suppurative parotitis, pseudo-diphtheria, etc. *Palpation* almost always shows an enlarged spleen, and not infrequently an enlarged liver. The vomiting which is usual at the onset is not due to the presence of pocks in the stomach, but to a catarrhal condition of the organ. Constipation is common, but diarrhea is also sometimes met with, being excited by a catarrh of the large intestine, and is especially common in children. The pocks may, however, be found in the rectum, where they sometimes excite dysenteric symptoms.

(f) **The Nervous Symptoms.**—The chief of these have been already pointed out, as well as the fact that complications and sequelæ are by no means rare. Violent delirium (previously referred to) may be followed by fatal coma, and in children convulsions may be seen. Very rarely paraplegia has been observed during the attack, though it is more common during the convalescence, and is then due to different causes, such as peripheral neuritis and disseminated myelitis (Westphal). Multiple neuritis may be a sequel or the pharyngeal nerve may alone be affected. Among other conditions rarely arising during convalescence are insanity, epilepsy, and hemiplegia.

(g) **The joints** may be swollen and painful after small-pox, the condition being secondary and rheumatic in nature, and in rare cases periorchitis may be observed.

(h) **Renal Symptoms.**—Apart from the febrile albuminuria already referred to, renal complications are of great rarity, and, with one exception (hemorrhagic nephritis), are of little practical import. Hemorrhagic nephritis may occur, and is always of a serious character, though, fortunately, it is very rare.

(i) **The Special Senses.**—The pustules may form upon the conjunctivæ and eyelids, and several important conditions result from this variolous

involvement of the eye—viz. conjunctivitis, keratitis, choroiditis, and panophthalmitis. Hebra met with ocular complications in 1 per cent. of 5000 cases of small-pox. Otitis media has already been mentioned.

**Special Clinical Forms.**—There are two unusual types of variola that are important in being severer than the moderate (discrete) form already described.

(a) **The Confluent Form.**—This is the result of an abnormally severe infection, but cannot be said truly to be atypical. The ushering-in symptoms are very severe, and the eruption may appear as early as, or even before, the third day, when the temperature remits, though not to any great degree. The separate papules are, perhaps, somewhat smaller than in the discrete variety, and vastly more abundant and close-set; and after the stage of pustule is reached the face and hands present an uninterrupted area of suppuration. The deformity of the countenance is correspondingly pronounced. Naturally, the local symptoms are intense and the fever and its concomitants are in exact proportion. The nervous symptoms often predominate. Salivation is frequent, and vomiting is excessive in adults, while diarrhea is equally so in children. The eruption may also entirely cover the mucous surfaces. The lymphatics of the neck may be greatly swollen—a circumstance that contributes to the patient's unparalleled disfigurement. The various complications previously adduced are of comparatively frequent occurrence, and following these a general pyemic process may then develop. When death occurs it is usually preceded by the development of the symptoms that belong to the typhoid state (typhomania, tremors, subsultus tendinum, a rapid, feeble pulse, dry, brown tongue, and diarrhea). On the other hand, if recovery ensues, it is tardy and often interrupted by complications and sequelæ.

(b) **Black Small-pox.**—In this form the blood is much changed, so that hemorrhages into the skin, mucous membranes, and various viscera occur. It is important to distinguish several sub-varieties, as follows: (1) A *benign* form, in which blood is infused into the pustules when patients are allowed to leave their bed too early in convalescence. Here the condition is due to the effect of gravitation, and hence is confined almost solely to the lower extremities. (2) Doubtless the ordinary variolous eruption may become *slightly hemorrhagic* without aggravating the constitutional condition. Often this is seen in debilitated and intemperate subjects. (3) In the same class of subjects a *dangerous hemorrhagic* tendency may be manifested. During any of the eruptive stages—papular, vesicular, or pustular—hemorrhages may occur into the eruption, and, moreover, free bleedings may take place from the various mucous surfaces (hematuria, epistaxis, hematemesis, enterorrhagia, etc.). The initial symptoms are usually intense, the eruption abundant, and in consequence of the hemorrhages collapse often occurs. The most serious complications, pneumonia, diphtheria, and nephritis (followed by uremia), are also apt to develop and terminate life, and even should recovery ensue it is very tedious. This and the subsequent sub-variety are truly anomalous. (4) A not uncommon form of hemorrhagic variola is met with in which the *acute hemorrhagic diathesis* develops during the period of invasion. Its onset is characterized by the usual symptoms intensified, and as early as the second day ecchymotic patches appear upon the skin-surface and grow rapidly by peripheral extension, the mucous surfaces also showing more



or less extensive ecchymoses. The regular variolous eruption rarely appears, though occasionally shot-like papules may be detected here and there. The temperature may be slightly elevated, but is rarely high, and not infrequently it may remain normal or subnormal. Death often occurs before the time for the appearance of the characteristic eruption, and very rarely does the patient survive the fourth or fifth day.

There are also varieties of small-pox that pursue an abnormally mild course. Of these (*c*) **varioid** deserves first place. By this term is usually meant small-pox occurring in individuals who have been protected by a successful vaccination, but it may also be the result of natural insusceptibility. Hence variola and varioid are one and the same affection, the latter name, however, representing a milder form of the disease than the former. The initial symptoms of varioid do not differ either in character or severity from those of true variola, but the general course of the attack is peculiarly prone to manifest irregularities. In the pre-eruptive stage an erythematous rash is very common, and its appearance is regarded by many as being of value in discriminating varioid from variola.

When the regular eruption appears the fever falls to normal and remains there. The rash comes out by the end of the first or on the second day, the papules being scanty, and quite as liable to appear first upon the trunk as upon the face. They are identical with the papules of variola, as is true also of the vesicles; but pustulation rarely develops, for the reason that resolution takes place, as a rule, before the latter stage is reached.

The secondary fever is either very slight or entirely wanting. The mucous surfaces are affected but to a correspondingly slight degree, and thus the general course of the disease is characterized by irregularity. Papules and vesicles may be found in close proximity; not so in variola. Desiccation begins between the fifth and seventh days of the eruption (the eighth and tenth of the disease), and hence, as compared with variola, the course is cut short and serious complications almost never occur.

(*d*) An **abortive** form is occasionally observed. It is characterized by the great intensity of the invasion symptoms, but these promptly subside, and the patient enters at once upon a stage of speedy recovery.

An exceedingly mild type may arise during seasons of epidemic prevalence of the disease, either with or without a scanty and undeveloped eruption, when the diagnosis is made entirely from the etiologic circumstances.

**Diagnosis.**—With a clear history and the presence of the characteristic features a positive diagnosis is a simple problem. But at any period before the papules are fully developed it may be confounded with certain other acute infectious diseases, notably cerebro-spinal meningitis, pneumonia, typhus fever, scarlatina, and measles.

**Differential Diagnosis.**—In *typhus fever* the onset is very like that of small-pox. The former may, however, be distinguished by its peculiar etiologic factors, especially its origin by importation or its non-prevalence in the vicinity; the appearance of the eruption, first upon the trunk (chest and abdomen) in the form of maculæ, and later becoming petechial; and by the fact that it is neither papular, vesicular, nor pustular, as in small-pox. Moreover, in typhus the temperature does not remit with the appearance of the eruption, but persists, and may even rise higher.

From hemorrhagic small-pox typhus is sometimes distinguished with great difficulty. In the most virulent type of the former death often



occurs before the eruptive stage is reached. When it does not, the causal data are most important factors in making a discrimination. In typhus shot-like papules are never detected upon the skin-surface of the hands and head, whereas they are sometimes found in hemorrhagic small-pox.

The diagnosis from *scarlatina* may early be made from the erythematous (scarlatinous) rash which often precedes the appearance of the variculous eruption. This is, as a rule, neither so intense nor so uniformly distributed over the skin-surface of the body as in true scarlatina.

*Lobar pneumonia* begins with many of the symptoms that characterize the initial stage of small-pox, but in addition there are sharp pain in the side, cough with rusty sputum, acceleration of the respirations out of proportion to the temperature and pulse, and the cheeks are bedecked with the typical mahogany flush.

*Meningitis* may be eliminated if the patient has been exposed to small-pox, and if, being unprotected by vaccination, he suddenly develops the symptoms of the initial stage of this disease—severe rigor with high fever and intense pain in the loins. To confirm the discrimination from meningitis we may note the absence of involvement of the ankles and other joints, the irregular temperature-curve, the herpes and opisthotonos, and marked hyperesthesia.

The *macular stage* of the eruption may be confounded with *measles*. The absence of the characteristic prodromes and symptoms of invasion belonging to the latter disease, the redness and swelling of the conjunctivæ, the photophobia and marked coryza, the stubborn cough, and increased fever after the eruption appears, make the separation easy. In measles the maculæ do not develop into hard, shot-like, conical papules as in variola.

Nothing, however, could be more difficult than to differentiate certain mild cases of discrete small-pox (in the non-vaccinated) and varioloid from *varicella*. In the table below, however, may be found a few contrasted points of distinction, which, I trust, may prove helpful:

VARIOLA.	History.	VARICELLA.
Absence of previous attack.	Same.	
Previous or present case in the vicinity.	Traceable to previous or present case of varicella.	
Not successfully vaccinated.	Negative.	
Occurs at any age.	Almost always in childhood.	
Characteristic pre-eruptive stage—rash on the third day.	Eruption not preceded by prodromes.	
	<i>Eruption.</i>	
Appears first upon the forehead, extending downward.	Appears first upon the neck and trunk—no regular progression over the body.	
Vesicles uniform in size, umbilicated, and deeper seated.	Vesicles vary much in size, are rarely umbilicated, and are more superficial.	
Eruption contains serum, later pus.	Only serum.	
Most abundant on face and fingers.	Most abundant upon back and lower extremities.	
Various stages of eruption observed at points removed from each other.	Various stages side by side.	
Pin-prick does not cause collapse of vesicles, being multicellular.	Does cause collapse, being unicellular.	
	<i>Secondary Fever.</i>	
Usually present.	Absent.	

**Prognosis.**—The prognosis depends upon (*a*) the degree of severity of the type, whether mild or intense, the severer forms (confluent and certain of the hemorrhagic) being grave. The hemorrhagic variety, in which large cutaneous ecchymoses suddenly develop, is almost invariably fatal, and often before the cases have advanced to the eruptive stage. The aggregate number of pocks that appear and the gravity of the infection are, as a rule, proportionate.

(*b*) The prognosis is modified by *individual peculiarities* (age, race, intemperance, etc.). Thus it is much more fatal in the very young than in older subjects, much more fatal in dark- than light-complexioned races, more fatal in the intemperate than in the temperate, and so on.

(*c*) Complications increase the death-rate considerably. Of these, broncho-pneumonia, lobar pneumonia, acute nephritis with uremia, septico-pyemic conditions, pseudo-diphtheritic angina, and pericarditis are most potent for evil. Among the foremost serious symptoms may be mentioned excessive vomiting, wild delirium, coma, a temperature of 106° F. (41.1° C.) or over, urgent diarrhea, and dysentery.

The death-rate has been computed to be between 15 and 30 per cent., varying, however, with each epidemic, and thus rendering an exact estimate out of the question.

**Treatment.**—The varied indications in the treatment of small-pox will be considered separately:

(1) **Prophylaxis.**—The rules that have been laid down elsewhere (*vide* Treatment of Typhoid Fever) for disinfection in infectious diseases must be rigidly enforced in this affection. Quarantine (*public and private*) must be secured if the deadly progress of small-pox is to be averted, and it would seem altogether unnecessary to adduce arguments to show the correctness of this dictum. In the homes of the poorer classes, however, and even in those of the higher, absolute isolation cannot be carried out successfully, and in view of this fact special, well-equipped hospitals should be provided for the reception of the disease. Without a rigid enforcement of these hygienic rules the spread of small-pox cannot be prevented. It is important also to remember that persons who have been afflicted with the disease cannot, with safety to others, resume their former places, either in the family or in society at large, before they are completely convalescent. The best means of prevention, however, is vaccination, and this subject will receive separate consideration (*vide* p. 229).

(2) **General Management.**—Apart from perfect isolation and thorough disinfection, there are other hygienic requirements that must be complied with. The room occupied by the patient should be large and freely ventilated (an essential matter, though strong drafts are to be avoided), and all carpets, curtains, and articles of furniture not absolutely needful should be removed.

The *diet* is an element of treatment that demands most careful attention, and should be varied according to the stage of the affection. During the initial stage it must be restricted to liquid nourishment (milk, animal broths, etc.), and in addition cooling drinks, including ice, lemonade, and other of the various fruit-juices (diluted). During the stage of remission of fever we may add soups, jellies, eggs, toast, etc., and with the onset of the stage of suppuration a supportive diet, reinforced by the judicious use of stimulants, is a highly essential part of the treatment. Light forms



of nourishment must now be given in definite quantities at short intervals, and stimulation carefully carried forward in accordance with the rules that ordinarily govern this class of affections.

(3) **The fever and associated symptoms** during the invasion stage are best controlled by the cold or gradually cooled baths, which possess all the advantages in this disease that they command in typhoid fever. Their effectiveness, together with the fact that in some of the various forms of variola the temperature-chart registers a normal or even a subnormal grade of body-heat, shows the fallacy of regarding temperature as the sole, or even the chief, indication for the use of the Brand method. Cold sponge-baths, the ice-cap, or the cold pack may be resorted to if cold immersion baths are not accessible to the patient. The internal antipyretics must be given with a sparing hand, if at all, and should be employed as antiseptic agents rather than as direct antipyretics, on account of their depressing effect.

The therapy of this stage also embraces the treatment of certain symptoms. The vomiting may be incessant and exhausting, and chipped ice, champagne, dilute hydrocyanic acid, and cocain hydrochlorate should be tried in the order mentioned. If diarrhea be severe, it should be checked (though neither wholly nor suddenly) by the use of arsenite of copper, the acetate of lead (gr. ij—0.1296) and opium (extr., gr.  $\frac{1}{4}$ —0.0162), in combination, or by bismuth salicylate (gr. v—0.324) and  $\beta$ -naphthol (gr. iij—0.1944). The nervous symptoms are usually restrained by the cold-bath treatment, but occasionally a wild delirium may necessitate further therapeutic interference, and at such times a combination of sodium bromid (gr. x—xv—0.648—0.972) with the deodorized tincture of opium (℥v—0.333), given every two or three hours, is of signal value. Very often the wise administration of stimulants removes all necessity for the use of further means of overcoming the nervous symptoms, and in maniacal delirium ether may be cautiously administered with great benefit. The catheter must be used if retention of urine should occur. For the intense pains that belong to this stage no other remedy can be compared with morphin sulphate (gr.  $\frac{1}{8}$  to  $\frac{1}{4}$ —0.008 to 0.016), to be administered hypodermically, and repeated if necessary; this measure also ensuring good sleep, which would be otherwise impossible.

(4) As previously stated, the **eruption** appears with the termination of the initial febrile period, and deserves the closest attention. The indications are twofold: (*a*) to limit the eruption as far as is possible, and (*b*) to modify its course, so that extensive suppuration and consequent disfigurement may be prevented. Ablutions with lukewarm water, to which may be added some antiseptic (carbolic acid and glycerin, or, better, a mercuric-chlorid solution—1 : 5000 or 1 : 10,000) will be found of great use. To prevent pitting many local applications have been used. Formerly, a common mode of treatment was to open the pustules as early as possible and touch them with silver nitrate—either in the solid stick or brushed over in a strong aqueous solution. Painting the skin with the tincture of iodine was a practice frequently followed in the past. The formula of Schwimmer, herewith given, gave excellent results in a case of my own:



R. Acid. carbolicæ,	4.0-10.0 ;
Ol. olivæ,	40.0 ;
Cretæ præparat.,	60.0.
M. et ft. pastamollis.	

Another serviceable combination is one of equal parts of carbolic acid and glycerin, to be applied only to the pustules. It has also been recommended to touch each pustule with carbolic acid, and then to apply this agent in equal parts with the oil of thyme (Sansom). It is important that only a certain proportion of the pustules be touched at once, this to be followed by an equal number on successive occasions. The parts must be kept aseptic and clean, while irritation from scratching, etc. must be carefully avoided. Moore of Dublin and Fingen have recently recommended the use of red curtains or shades to cut out certain chemical rays. This treatment was first practised by John of Gaddsdén, a court physician of the fourteenth century.

During convalescence, or as soon as the general condition of the patient will admit of it, warm baths, with the free use of carbolic soap, are to be given at intervals of two days until several baths have followed the separation of the crusts. Any cutaneous sequelæ that may present themselves must be attacked in accordance with ordinary principles.

(5) **The Period of Remission of Fever.**—There are very rarely any symptomatic indications apart from those presented by the eruption. It is of first importance, however, to thoroughly support the powers of the system by means of tonics, and especially by quinin, in addition to an appropriate diet and the moderate use of stimulants.

(6) **The Suppurative Stage.**—All measures tending to support the strength of the patient are needed—the mineral acids, with the elixir of calisaya, quinin, strychnin, etc. Stimulants are often required, and it may become necessary to give them unsparingly, the character of the pulse and of the first sound of the heart, as well as the nervous symptoms, being the physician's guides. Gradually cooled baths of the usual duration or warm baths somewhat more prolonged give excellent results. In this stage certain symptoms may require special treatment. The ulcers in the mouth and throat are best relieved by the use of a saturated solution of chlorate of potash in water as a gargle or in the form of an atomizer spray. Ice allowed to melt in the mouth is also valuable. Hemorrhages demand prompt interference, and full doses of ergot must be given subcutaneously. Internally, large doses of the tincture of the chlorid of iron, gallic acid, the mineral acids, or turpentine may be administered.

The complications, as before intimated, are not numerous, and are for the most part secondary in nature. Those connected with the respiratory passages should be prevented if possible, and, owing to the fact that they indicate danger, should receive active treatment if they occur. By frequently changing the position of the patient when bronchitis is present, and by encouraging him to cough frequently, as well as by the timely use of stimulants and the proper care of the mouth, pulmonary complications can often be obviated. Should lobular pneumonia occur, the plan of treatment which is likely to meet with most success may be briefly put thus: Free stimulation, the assiduous use of the cold or gradually cooled

baths, tonics, and nourishing foods. *Laryngeal perichondritis* with edema of the glottis may suddenly demand tracheotomy. To avoid the development of *bed-sores* an air-cushion or a water-bed should be provided, if needful. Care should also be exercised to prevent ocular complications, and their occurrence demands a vigorous form of supportive treatment. I have much confidence in the use of cold compresses, instilling into the eyes at the same time a solution of boric acid (gr. x to xv—0.648 to 0.972—to f3j (30.0).

(7) **Special Modes of Treatment.**—These would be found to be numerous were we to enumerate all of them, but only those based on the principle of antiseptics are worthy of notice. According to one plan, which has many advocates, antiseptic agents are administered internally. The remedies that have been most frequently employed in this manner, and with perhaps the most promising results, are the sulphocarbolates, salol, sodium salicylate, carbolic acid, creasote, mercuric chlorid, and the sulphites. Zuelzer states that xylol given internally is potent in coagulating the contents of the pustules, but experience does not corroborate this opinion.

Kinyoun, Lundmann, and Bécclère have used the serum from vaccinated subjects (human beings and the lower animals) or from variolous patients in advanced stages of the disease in the treatment of small-pox. The cases, however, are insufficient to warrant deductions.

**Special Methods of External Medication.**—Dr. Galewouski<sup>1</sup> reports brilliant results in the treatment of variola with baths of potassium permanganate. The salt is added till the water is of a rose-red color, and Galewouski claims that by its application the temperature is lowered, the pustules disappear, and recovery speedily ensues. Talamon<sup>2</sup> recommends a special plan of external medication in the form of a mercuric-chlorid spray for small-pox vesicles and pustules, his object being to keep the surface under the influence of an antiseptic. The mercuric-chlorid solution is prepared after the following formula:

R <sub>x</sub> . Mercuric chlorid,	gr. xv (1.0);
Tartaric acid,	gr. xv (1.0);
Alcohol (90 per cent.),	f3jss (6.0);
Ether to make	f3jss (45.0).

Sig. To be applied as a spray three or four times daily for one minute.

It is essential to exercise the precaution to protect the eyes, which may be covered by layers of cotton dipped into a saturated solution of boric acid. Talamon advises the commencement of his method on the first day of the eruption, the application to be preceded with a vigorous washing of the face with soap, which may be rinsed off with boric acid and then dried with absorbent cotton. After the spray has been used the face should be covered with a layer of a 50 per cent. glycerolate of mercuric chlorid in order to keep the skin continuously aseptic. After the fourth day the number of sprayings per diem is gradually lessened, so that by the seventh day they may be discontinued; but the application of the glycerolate should be continued.

<sup>1</sup> *Med. Press and Circular*, 1890.

<sup>2</sup> *Journal of Cutaneous and Venereal Diseases*, February, 1891.

Talamon added, in the confluent and other grave forms of the disease, general mercuric-chlorid baths, lasting for three-quarters of an hour to an hour. The buccal and pharyngeal eruption is to be treated by gargles and lotions of boric acid. Internally, the therapy is limited to sustaining the strength of the patient by means of alcohol (3ij-iv; 90.0-120.0 daily), according to the gravity of the case.

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## VACCINATION.

**Historic Note.**—One of the first steps in preventive medicine was the practice of inoculation as a method of protection against the infection of small-pox. It had been practised in China and other Asiatic countries for centuries, and Lady Montague, the wife of an English ambassador to Turkey, early in the eighteenth century introduced it into England, after which time and until vaccination was known, it was very extensively practised there.

Pus taken directly from a small-pox pustule was introduced beneath the epidermis, and the person inoculated developed variola, though in a milder form than when arising from ordinary infection. The attack ran a more rapid course, having fewer pustules, rarely terminating fatally, and protected the individual from subsequent attacks.

The objections to this method were that it did not always produce a mild form of variola, a small percentage of cases having a fatal termination, and that, however mild the attack, other unprotected persons brought in contact with it were as liable to contract small-pox in as virulent a form as if contracted in the usual way.

In a paper published in 1798, Edward Jenner, a physician of Gloucestershire, England, and a pupil of John Hunter, first made known to the world the value of vaccination. Twenty years previous he had observed that persons employed in dairies, who were accidentally inoculated with cow-pox, were insusceptible to the contagion of small-pox, and, after experimenting all these years, he became satisfied that inoculation with the vaccine lymph was a preventive against small-pox. After the publication of his paper he was subjected to ridicule and abuse by the profession, but through his persistence he was finally allowed to practise his method of vaccination in the wards of a hospital, and in the course of a few years it became generally recognized and was practised in France and America, as well as in England. Later, the method fell into disrepute for a time, owing to the fact that certain persons who had been vaccinated subsequently contracted the disease, it not being known then that a revaccination was necessary from time to time. Strange to say, however, in the century that has passed since the first vaccination by Jenner there has practically been no change or improvement either in the method or the vaccine used.

**Vaccinia, or cow-pox,** is a mild eruptive disease that occasionally occurs among cattle, a similar disease being produced in them by inoculation with the small-pox virus from man. It is communicable by contact only, and is usually carried from one cow to another by the hands of the milkers; hence being usually found on the udder or teats of milch



cows. Since Jenner's time many theories have been advanced as to the exact nature of this disease in cattle, and at the present day the subject is still in dispute. It is now, however, generally conceded that if cow-pox is a distinct disease, originating only with the cow, the eruptive disease produced in this animal either by inoculation of small-pox virus from man or of "grease" from the horse is, at least in all essential respects, a disease not to be distinguished from primary or idiopathic vaccinia. Guarnieri has described certain parasitic organisms, the *Cytorectes Guarnieri*, found in corneal lesions produced by the injection of vaccine lymph. This observation has been confirmed by Pfeiffer and others, but the pathogenic nature of these protozoa has not been determined.

The *vaccine virus* consists either of the liquid contained in the vesicle or of the scab resulting from the desiccation of the pustule. The former is furnished from vaccine farms, of which there are several in this country, is then dried on ivory points, and, if kept in a cool place, retains its virtue for a week or ten days, or, possibly, longer, but should be used as fresh as possible to ensure a successful result. It is also sometimes preserved in capillary glass tubes, sealed at both ends, or between glasses, and kept in this way it is less liable to infection through uncleanness in handling. The scab from the cow is not used.

*The Site.*—The point usually chosen for vaccination is on the arm over the insertion of the deltoid muscle; but in girls, for cosmetic reasons, it is sometimes preferred on the leg, and the most common site is over the junction of the two heads of the gastrocnemius muscle.

*Technique in Vaccination.*—The part selected should be made surgically clean; then gently scrape the skin with a lancet or other instrument, which has also been made aseptic, until serum begins to exude. If, by too vigorous scraping blood should be drawn, it should be carefully dried with a piece of sterile cotton before the lymph is applied.

The charged end of a point, which has been previously dipped in tepid water, is now gently rubbed over the abraded spot and the limb left exposed to the air until the lymph has been dried upon it. It may then be protected by a piece of gauze strapped on it or by one of the shields made for the purpose.

Some physicians still prefer the *humanized lymph*, and when this is used the "arm-to-arm" vaccination is best. The lymph is taken from a characteristic vaccine vesicle (from the fifth to the seventh day of its development) of a healthy child and applied directly to the arm of another. When this method is not practicable, however, the virus may be dried and preserved for use just as in the case of bovine virus.

The *scab* resulting from a vaccine vesicle on a healthy child was formerly quite generally used, and it could be kept a long time without losing its virtue. It was sure in its action, and offered the advantage to the physician of being easily preserved; but it was more liable to become infected than the lymph when preserved in the usual way, and, since the vaccine farms are so conveniently located, lymph may be obtained from them at any time without delay.

There are no valid reasons why the humanized should be preferred to the bovine lymph, and the possible danger of conveying syphilis or other constitutional disease from one person to another by means of the former should be sufficient reason for the use of the latter.

*Period of Life for Vaccination.*—It is usually advised to vaccinate infants within a few weeks or months after birth, but, unless small-pox is prevalent, it is best to wait until the latter part of the second or the beginning of its third year, as the child has then passed through its teething period and will be better able to resist the effects (slight though they may be) consequent upon vaccination.

*Time for Revaccination.*—To ensure the individual against infection he should be revaccinated at puberty and every few years afterward, or at any time when small-pox is epidemic or liable to become so.

**Symptoms.**—After vaccination no local or constitutional effects—except the slight irritation due to scarification—are noticed until the third day, when a small red papule appears. By the fifth or sixth day a vesicle appears. By the ninth day it is fully developed, and, like the vesicle of variola, is filled with colorless lymph, is umbilicated, multilocular, and has a distinctly inflamed areola of deep red color, accompanied by heat, itching, and tenderness. By the tenth day this may extend an inch or two from the vesicle. Quite frequently the axillary or inguinal glands (depending upon the location of vaccination) are swollen and tender, and in a tubercular child they may go on to suppuration. After the tenth day all these symptoms gradually decline; the pustule dries up, and then forms a brown scab which is usually detached in the third or fourth week, leaving a permanent cicatrix.

**Complications.**—Occasionally one or more additional vesicles are formed at a little distance from the point of inoculation, and, rarely, there is a general vesicular eruption, due to absorption of the lymph. An *erythematous rash* is not uncommon, and appears, if at all, about the sixth day. *Erysipelas* may occur as a complication, and, if it is prevalent in the house, vaccination should, as a rule, not be performed; if deemed necessary, however, the greatest care should be taken to ensure cleanliness.

Sometimes, owing to injury to the vesicle or to uncleanness, an ulcer forms, which may be weeks in healing. *Eczema* and other skin-affections are usually aggravated during the course of vaccination, and it should not be forgotten that it is possible for *syphilis* to be inoculated with the vaccine virus. Any of these complications call for the same treatment as when occurring independently.

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## VARICELLA.

(*Chicken-pox.*)

**Definition.**—An acute, contagious disease, characterized by a cutaneous eruption of papules, passing into vesicles and pustules; also by slight fever and mild constitutional symptoms. For a long time it was confounded with varioloid, but its distinct character has now been recognized for many years. Complications and sequelæ are infrequent.

**Etiology.**—It is well established that the contagium of varicella is found in the vesicles, as the disease has been communicated by actual



inoculation with their contents. The specific poison has not been satisfactorily isolated, although it is suspected that certain protozoa are the direct cause, but, as in the case of vaccinia and small-pox, positive proof is wanting. Varicella may be transmitted by exposure to another case or possibly through the medium of a third person, the school and asylum being the most frequent points of its origin. It affects children of all ages, and usually one attack is protective. It closely resembles measles in its contagiousness.

**Symptoms.**—The *incubation* period is uniformly from fourteen to sixteen days. If there be a prodromal stage of the disease, certainly in the vast majority of cases it cannot be recognized, though a slight fever and general indisposition may be noticed for twenty-four hours before the appearance of the eruption. In many cases the eruption is the first symptom. This occurs in the form of small reddish puncta, from which rapidly develop rosy-colored maculations, and these become tensely distended, transparent, or slightly yellowish vesicles of the average size of a split pea. The eruption appears first upon the upper part of the body, the chest and back, neck, scalp, and face (on the latter quite sparingly), and always upon the hairy scalp. Frequently the vesicles form on the mucous surface of the lips, inside the cheeks, on the tongue, palate, conjunctivæ, and in the progenital regions of both sexes. At times the glands of the throat become slightly enlarged and painful, the vesicles are superficial, the child has the appearance of having received a shower of boiling water, and the firm papule which precedes the variolous rash is altogether wanting. The vesicles are at first transparent, and their contents plainly show through their translucent roof-wall which is composed only of the stratum corneum of the epidermis. Umbilication rapidly occurs at the apex, and the contents of the vesicles become lactescent, and gradually sero-purulent. The areola is most distinct when the vesicle is fully formed and fades as the latter dries. Crusts form which drop off in from five to twenty days, depending upon the depth to which the skin has been involved. On the trunk, as a rule, no mark is left, but after the more severe attacks, when the true skin has been involved, scars remain, and frequently there is quite deep pitting. The marks are usually on the face when the skin has been unprotected. On the hands and feet the vesicles appear without having been preceded by a papule, and sometimes there is no areola, each vesicle resembling a drop of water upon a healthy skin. Pustules may develop in consequence of irritation or infection, as the result of scratching, or in feeble or poorly-nourished children and in unhealthy children deep ulceration may occur, lasting for weeks. In rare cases there may be necrotic inflammation about the site of the pox (*varicella gangrænosa*).

In mild cases only ten, twenty, or thirty spots may be found on the body, but in severe cases the skin may be almost covered in certain regions. The eruption, however, is never confluent. The temperature is highest on the second or third day, when the eruption is appearing. In mild, uncomplicated cases the thermometer registers 101° or 102° F. (38.8° C.) for two or three days at most, but in severe cases the temperature may be as high as 104° F. (40° C.). This is usually due to broken health prior to the acute illness. The temperature falls gradually as the rash fades, and presents a temperature-curve similar to that of measles.



There is usually neither coryza, vomiting, cough, nor diarrhea, and in their place is only the general indisposition which is associated with any febrile disease.

**Complications.**—*Erysipelas* occasionally acts as a serious complication in delicate children. It may develop about the pocks, particularly when they are deep and associated with some ulceration, and scratching with unclean fingers is its prime causal factor.

*Adenitis*, mild and isolated, and *suppuration* with abscesses in the deeper cellular tissue are occasionally seen.

*Nephritis* is infrequent, but may occur in unhygienic surroundings or in carelessly managed cases, just as it may follow scarlet fever or measles.

Varicella is also quite frequently complicated with other infectious diseases, and varicella, scarlet fever, and measles have been seen curiously blended in epidemic form. Varicella and measles, however, are most frequently associated.

The **diagnosis** of varicella offers no special difficulties. The eruption comes out slowly and in crops, so that papules, vesicles, and crusts may be seen upon the skin in close proximity. Again, it should be noted that the umbilication is due only to the fact that the drying up of the vesicle begins at the center, and that the pocks may appear on the mucous membrane. Varicella is distinguished from *urticaria* by the presence of fever, and from *eczema pustulosum* by the mild febrile symptoms of the latter, the discreteness of its pustular lesions, the absence of itching and of infiltration of the skin in patches, and by its tendency to symmetric development.

*Variola* and *varioid* of infants are to be distinguished from varicella by the prodromal symptoms, and by the greater rise of temperature, though the distinction between mild varioid and severe varicella in infancy and childhood will always tax to the utmost the skill of the keenest diagnostician. The sooner it is understood that intermediate forms are likely to occur, which cannot be positively assigned to one or the other category, the better it will be for both the profession and the laity.

The **prognosis** in private practice is always favorable. Only in the slums or in hospital cases complicated by *erysipelas*, *adenitis*, or *nephritis* may grave results be anticipated. The milder cases may, however, leave slight monuments of their existence in the form of one or more depressed cicatrices which may mar an otherwise beautiful face.

**Treatment.**—Isolation should be enforced in schools and in all institutions containing many young children. In private houses, unless the younger children are delicate, quarantine is unnecessary. The disease may be transmitted to others as long as the crusts are present, and hence isolation should be maintained until they have fallen off. In most cases constitutional symptoms of the disease are so mild as to require no treatment. It is best at the outset to place the child in bed for a few days, and sponge daily with warm carbolyzed water; the local itching may be allayed by sponging with a weak solution of carbolic acid or by the use of carbolyzed vaselin. When the crusts have formed, especially on the face, an ointment of zinc oxid containing ichthyol (2 per cent.) should be applied, and care should be exercised to keep the skin clean and to prevent scratching. In all cases the urine should invariably be examined several times during and following the attack.

## SCARLET FEVER.

(Scarlet Rash; Scarlatina.)

**Definition.**—Scarlet fever, or scarlatina, is a self-limiting, acute, contagious disease, characterized by vomiting, fever (more or less typical), angina, and in twelve or twenty-four hours by a diffuse, punctiform, scarlet eruption, followed by membranous desquamation and, frequently, by nephritis. It is a disease of childhood, but may occur at any time of life.

Scarlatina is a widespread disease, though perhaps less universal than measles. It is endemic in all the large cities of the globe, and at intervals the cases multiply into more or less extensive epidemics. Smaller towns and rural districts are visited, and the epidemics are usually traceable to importation of scarlatinal poison, so that it may be stated that they never originate *de novo*.

**Pathology.**—There are no pathognomonic changes. When death occurs early the chief lesions are presented by the throat, while in addition engorgement of the viscera is noted, especially of the brain. The exanthem is rarely visible. In malignant types, however, in which the eruption is not seen during life, it makes its appearance rarely after death, and this aids in establishing the nature of the affection.

When death occurs at an advanced stage the lesions are those either of nephritis (with dropsy), or of septicopyemia, or of inflammation of one or more of the serous surfaces (pleurisy, pericarditis, endocarditis, meningitis, etc.). Additional changes in the various viscera are, for the most part, identical with those met with in other acute infective diseases, and hence need not be described here. The blood, it should be pointed out, is dark, fluid, and coagulates feebly, owing to a decrease in its fibrin factors. The process of desquamation may be observed, together with more or less emaciation in protracted cases.

Among other lesions which are more or less peculiar to the disease are (a) The *eruption*, which is a dermatitis of very mild grade. (b) *Scarlatinal angina*, which in its mildest form presents hyperemia and a slight swelling of the mucosa of the tonsils, soft palate, and pharynx. In the severer grades the inflammation is phlegmonous (scarlatina anginosa), and sometimes terminates in ulceration. There is great swelling (especially of the tonsils), and the formation of abscesses, due to secondary infection, is common. Extension of the purulent inflammation to the connective tissue of the neck produces marked induration, and more or less extensive abscesses may take place. Gangrene sometimes supervenes. (c) In certain epidemics a *membranous exudate* accompanies the *scarlatinal angina*, and this may or may not be truly diphtheritic. When it appears early it is non-diphtheritic, as a rule, the streptococcus of erysipelas being often found; on the other hand, when it comes on late it is often diphtheritic in nature and shows the presence of the Löffler bacillus. Other secondary inflammatory processes occur that are due either to direct extension or to metastasis, but these will be considered later at sufficient length (*vide* Clinical History). (d) *The Nephritis.*—The renal lesions, so prominent and so common in scarlatina, are included in the description of "Acute Bright's Disease."

**Etiology.**—The **bacteriology** of the affection is unknown as yet, although the fact that it depends upon a special poison cannot be doubted.



As early as 1892, Doehle described a peculiar variety of protozoa which he found in the blood of patients suffering from some of the eruptive fevers, and among them scarlet fever. He found a ciliated and a contractile form, occurring both within the red blood-cells and free in the plasma, and staining well with methylene-blue. This observation, however, has not been confirmed, and as the streptococcus pyogenes has been found in nearly all the inflammatory complications of the disease, especially scarlatinal pneumonia and angina, some pathologists (Babés, Bergé, Klein) have held it to be the cause. Marmorek, Raskin, and Mosny, however, believe that it is an example of mixed infection, the streptococcus being merely a secondary factor, and Marmorek has been confirmed in this view by the results of his experiments with antistreptococcus serum.

The general receptivity for scarlet fever is not so great as in certain other exanthemata (*e. g.* small-pox, measles); hence in a household in which there are several children some are apt to escape the disease, even though all have been equally exposed.

The virus is probably contained in the excretions from the throat and in the epidermal scales thrown off from the surface of the body. It is also present in the blood of scarlatina patients.

**Modes of Conveyance.**—The majority of the cases are produced by *contagion*, and I have observed that in many instances a single contact of a healthy child with a scarlet-fever patient has sufficed to convey the disease. It is also communicated by *fomites*, and the poison of scarlatina contained in clothing retains its infective power for months—a fact that shows conclusively its great tenacity of life. The patient himself is a center of infection until the end of the period of desquamation. Again, any objects (furniture, utensils, library books, toys, etc.) which the patient has touched or handled may serve to communicate the poison to healthy children. The disease may also be transferred by persons who have been in the sick-room, while they themselves escape. Transmission through milk has been observed, infected dairies having been known to disseminate the poison and give rise to epidemics. The infection may also be air-borne, though not for any great distance.

**Mode of Infection.**—The precise way in which the infection of a healthy person takes place is not quite clear. Most probably the poison is inhaled into the throat, where infection usually occurs, but it may gain entrance to the body through the alimentary tract. Infection may also take place through the blood, as is shown by the fact that children have been born in all stages of the disease. Artificial inoculation with the blood of scarlatina patients has resulted in more or less typical forms of the complaint. Whether or not an open lesion of the mucous membrane of the throat or other surfaces is necessary for the entrance of the poison into the circulation has not been determined positively, but fresh wounds always predispose to infection.

**Predisposing Causes.**—(1) *Age.*—The period of chief liability is from the second to the tenth year, after which it diminishes. It is rare under the age of one year, and especially so under six months. (2) *Recent wounds*—accidental or surgical—increase the susceptibility to the peculiar poison. (3) *Women in childbed*, for the same reason as (2), but care must be exercised, lest this class be confounded with septic affections. (4) *Season.*—The autumn and winter months furnish the most cases.



**Immunity.**—Single attacks during the life of a person form a rule to which there are rather frequent exceptions.

**Clinical History.**—The incubation period is extremely brief, lasting usually from two to three or four days. It may rarely, however, be longer—five to eight days—or more rarely still shorter—less than twenty-four hours.

The *invasion* of scarlet fever is generally quite sudden and, as a rule, active. The child feels uncomfortable, looks stupid, complains of sore throat and decided nausea, and in the great majority of the cases vomits. The tongue is furred. If he be very young, nervous symptoms are prominent, and he may exhibit convulsions. The pulse, which is a strong diagnostic factor, is rapid and hard, reaching 140 to 160 at the very onset. The temperature rises quickly to 104° or 105° F. (40.5° C.), and remains high.

**Eruption.**—Within the first twenty-four or thirty-six hours the characteristic rash appears, and is, as a rule, first seen on the neck; there is no certainty about this, however, as it may first come out on the abdomen or back of the hands or on the thighs, and not be seen on any other part of the body. Frequently it is found on the dependent portions of the trunk. At first it is slight, but perfectly characteristic, and usually takes two days to mature. In mild cases it disappears within thirty-six to forty-eight hours, and at no time is more than a very fine rash, but when typical it cannot be mistaken, especially if accompanied by the premonitory symptoms. When seen from a short distance at the end of the first twenty-four hours of its appearance the whole body (except the face) is of a uniform bright scarlet color. If we examine more closely, we find that the eruption consists of a multitude of red points that correspond to the hair-follicles. These points are surrounded by zones of erythematous redness, which, joining with one another, give a generally diffuse red appearance to the whole skin. Frequently, however, the rash consists of points representing the hair-follicles without the erythema, and in rough skins the rash may be more punctiform—that is, more strictly a condition of “goose skin.” Sudamina are quite frequent. Pressure by the finger causes a pallor which at once disappears when the finger is removed. The patient’s lips and chin are pale and in striking contrast with the vividly scarlet cheeks. In some cases the rash is patchy, especially on the limbs, and in these cases it may suggest measles, the patches consisting of clusters of fine papules or points with much surrounding erythema, while normal skin is present between the patches. In severe cases the rash may be hemorrhagic in character, minute extravasations of blood taking place in the skin; this may occur even in mild attacks, and not be seen until after death, but more frequently it is seen in malignant cases. Purpuric patches are frequently found after death when even in life they did not appear. There is itching, which may be either moderate or intense throughout the eruptive stage.

By the end of the first week the rash, which has been fading for several days, is succeeded by a desquamation that will be extensive or slight according to the intensity of the fever. In mild cases the tonsils, palate, uvula, and pharynx are deeply congested, and the mucosa of the cheeks, palate, and tonsils may show the eruption. In severer forms the tonsils are red and inflamed, and covered with tenacious secretions,

while minute yellow points corresponding to the tonsillar crypts are usually prominent. (*Vide* Malignant Scarlatina.) The nasal chambers are swollen, producing a free discharge, and the deeper cervical glands at the angle of the jaw are frequently enlarged. The *tongue* is coated with a thick, dense white fur (dead epithelium), and frequently shows a dry, glazed central band. In a few days the dead epithelium is cast off, clearing the tongue, when we have a red, clean, glazed tongue with greatly enlarged fungiform papillæ, giving us the *strawberry tongue* of classical history. The eyes are frequently swollen and the conjunctivæ injected.

Sleeplessness and mild delirium often mark a typical case, suggesting a congested state of the meninges, but it is neither usual for the child to be violent nor for the delirium to continue long.

The pulse is usually a strong diagnostic feature, and is always hard, quick, and wiry, varying from 140 to 160; it is out of proportion to the temperature and the general condition of the child. Leukocytosis is commonly noted. The temperature in average cases reaches 104° or

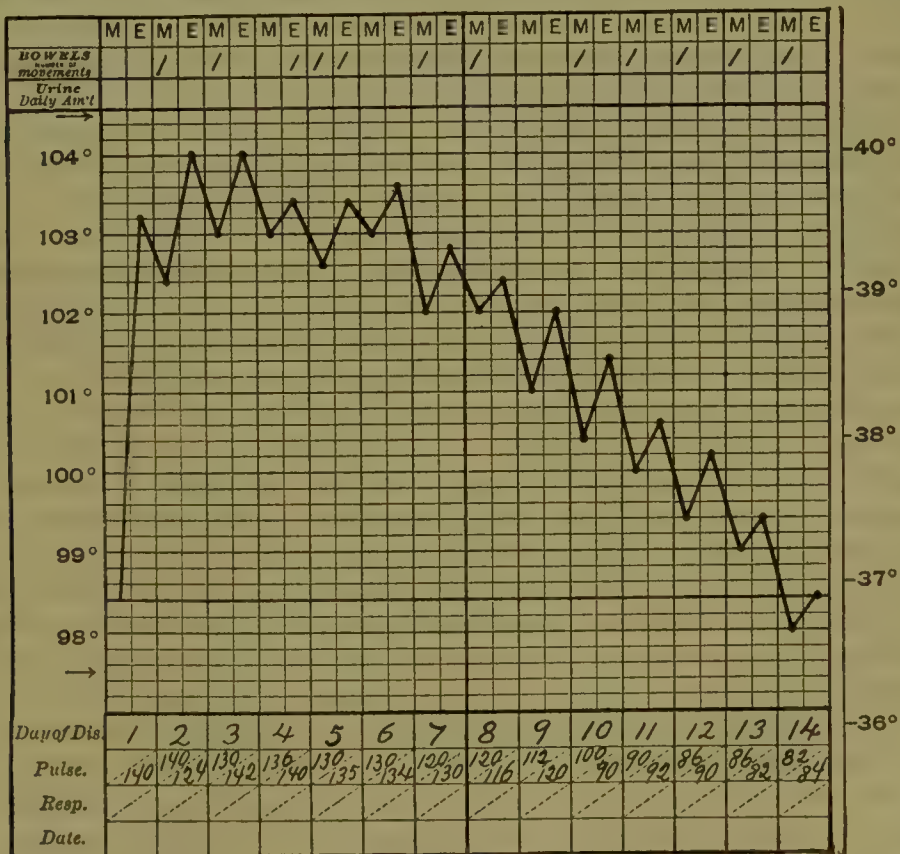


FIG. 22.—Temperature-curve of a case of scarlatina with favorable course—William C., aged seven years.

105° F. (40.5° C.), and in severe forms it may touch 106° F. (41.1° C.), the nocturnal remissions being slight and defervescence gradual (*vide* Fig. 22). The urine is scanty, thick, and contains urates, with a small quantity of albumin.

Within one week, if no complications have occurred, the attack will have reached its height and the symptoms have begun to decline. The



rash gradually fades, temperature falls, the tongue is less red, the throat less injected, and the child seems more natural. If at the end of one week the fever continues, it suggests the many possible complications, the most frequent of which is a throat or tonsillar ulceration, inflammation of the cervical glands, otitis, or, most probably, acute nephritis. It must be well understood that no two cases of scarlet fever are alike.

**Clinical Types.—Mild Scarlet Fever.**—In very many cases of scarlet fever all the premonitory symptoms are absent, and the rash is the only indication of the trouble. There is neither vomiting nor fever to be recognized, and no tonsillar trouble of any importance, while the rash is neither uniform nor well marked. In these cases we must be very careful not to confound the eruption with urticaria or some of the many medicinal rashes. The most difficult cases of all to diagnose are those in which sore throat is present without a rash, inasmuch as there is nothing characteristic about a scarlatinal tonsillitis.

During house epidemics when several children are affected it frequently happens that a child has sore throat and the “strawberry tongue” without a development of the rash. This may also occur in adults, and is the so-called *scarlatina sine eruptione*. These very slight cases of the fever are the most to be dreaded, as they may be followed by the most severe attacks of nephritis.

**Malignant Scarlet Fever.**—Death occurs usually by the end of the first week in severe cases, Drs. Ashby and Wright reporting a death within the first twenty-four hours (*atactic form*). In malignant cases, such as usually occur among the unhygienic and delicate, the tonsils may be covered by a membranous exudate, and the system quickly receive an overwhelming dose of the poison; death then results from septic causes (*anginose form*). In cases in which death occurs early a child soon becomes cyanotic, restless, or more frequently somnolent. In all these cases the temperature remains high— $105^{\circ}$  to  $106^{\circ}$  F. ( $41.1^{\circ}$  C.), and very frequently  $107^{\circ}$  F. ( $41.6^{\circ}$  C.). Diarrhea is frequently a troublesome factor in severe cases; coryza is very abundant; there is much glandular swelling and cellulitis, the neck becoming enormously enlarged and hard, the skin dull and livid in color; the extremities grow cold; the heart gradually becomes irregular, losing a beat, and finally fails.

If life is sustained through such an ordeal, the tonsils slough and the lungs may eventually become the seat of a septic pneumonia. In many desperate cases when life is prolonged to the end of the second or even third week general septicemia is most likely to occur. In this condition the tonsils ulcerate, sloughing patches appear on the fauces, the glands about the neck become enlarged and doughy, and the nasal mucous membrane gives out a purulent secretion in abundance. The temperature may remit, but continues high; the urine is albuminous; pus wells from both ears; and thus the child is gradually consumed and perishes. In all these cases pleuro-pneumonia will be found, together with hemorrhagic kidneys, and most probably small abscesses will be found in the latter at the post-mortem.

A third variety (*hemorrhagic*) shows at first cutaneous petechiæ, which grow rapidly into large ecchymotic patches. Hemorrhages also take place from the mucous surfaces, epistaxis and hematuria being very common. Death, as a rule, follows in two or three days.



**Desquamation.**—By the end of the first week the rash commences to disappear, the skin is (or soon becomes) mottled, dry, and rough, and gradually the scarf skin begins to separate. This process usually begins about the neck and trunk, and frequently large flakes are detached, the whole cuticle of the hand or foot sometimes coming off in one mass like a glove. The degree and character of the desquamation bear some relation to the severity of the eruption. In some cases the hair and nails have been cast off. In many cases desquamation is prolonged to the eighth week; it is usually longest on the hands and feet.

**Complications.**—**Otitis.**—The inflammation may extend from the throat along the Eustachian tubes to the middle ear, and pus be formed in the tympanic cavity, making its exit by perforating the membrane. This complication may occur either during the fever or at some time during convalescence. Suppuration in the middle ear is one of the common causes of a continued high temperature after the disappearance of the rash. Pain in the ear may not attract our attention to this unfortunate complication; most frequently, however, the child will place its hand on the ear and shake its head, as if to get clear of some source of irritation.

**Pyemia.**—Pyemia and abscess of the lungs may follow, and thrombosis of the lateral sinus may occur. The tonsils may be the seat of deep ulceration, and the soft palate may slough and show cicatrization of the soft parts of the throat in cases which may yet recover. The cervical glands may become enlarged and suppurate, either during the fever or while the child is convalescent. In debilitated or strumous children this complication may be very troublesome, with the formation of deep ragged ulcers, slow to heal, and in rare cases exposing the larger blood-vessels. Broncho- or lobar pneumonia may occur, and is most usual during the second week, being due to extension downward of the lesion from the throat. Pneumonia followed by empyema may also occur during convalescence.

**Arthritis, Synovitis, and Rheumatism.**—In unhygienic surroundings or where careful nursing is not carried out the joints are likely to become swollen and tender at the end of the first or the commencement of the second week. The wrist and the small joints of the fingers are most prone to be affected; the synovial membranes and the sheaths of the tendons at the back and in the palms of the hands are attacked. The elbows and joints of the vertebræ, the ankles, the knees, and soles of the feet may be affected; movement causes pain, and these parts are generally swollen, red, and very tender. The trouble is not severe, being fugitive and seldom returning to the same joint. Etiologically, this form of trouble is not true rheumatism, but is analogous to gonorrheal synovitis. The knees may be most severely affected, remaining swollen for several weeks, and in unusual cases suppuration may take place and be followed by pyemia. Such cases are usually fatal.

**Nephritis.**—No other complication of scarlet fever can equal nephritis in importance or interest, this condition always giving rise to anxiety in otherwise mild and hopeful cases. During the height of the fever, as is the case in all exanthemata, there is a trace of albumin in the urine that has no special significance, and it is possible for the kidneys to escape without greater damage than occurs in other acute febrile affec-

tions. Independently of this febrile albuminuria, there are two forms of nephritis which it is important to bear in mind, though they have been frequently confounded:

(a) *Septic Nephritis*.—In severe forms of scarlet fever, when the throat symptoms include sloughing tonsils, involvement of the soft palate, and general adenitis, the urine quickly becomes loaded with albumin, but shows scarcely any blood and but few casts. No renal symptoms will be recognized, and if present they may be masked by the general condition of septicemia. There may be neither dropsy nor uremic phenomena, but the patient usually dies by the end of the second week, when a typical pyemic kidney is found containing minute abscesses. This condition of the kidney is only one part of the general pyemia, and merely illustrates the fact that this organ suffers during the course of the general inflammation.

(b) *Post-scarlatinal nephritis* is the form most likely to occur about the third or fourth week, and is the one generally known as scarlatinal nephritis. The kidneys are undoubtedly involved in an active sympathetic inflammation, and at the end of the fever, more than at the beginning, are engaged in carrying off waste products of the fever itself. From the nature of the disease they are in an irritable condition and prone to take on inflammatory changes, just as the bronchial tubes and the lungs are left in a very susceptible condition following measles and whooping-cough. In this way the uriniferous tubules become choked up by the desquamation that is going on inside. The number of cases that suffer from post-scarlatinal nephritis varies according to social conditions, the nature of the epidemic, the season of the year, the nature of the treatment received during the disease, and especially the care received throughout convalescence. Ashby and Wright fix the rate of those who suffer at 6 per cent. of hospital cases, but this is, undoubtedly, too low, since hospital cases receive better care during convalescence than private cases. The usual time for this form of nephritis to occur is from the end of the second up to the fourth week, but it usually begins very insidiously. Traces of albumin may be found for a few days before the blood and larger quantities of albumin occur, but it is often impossible to date the commencement of an attack. Usually after the fever has subsided the patient for a few days feels well, but very suddenly grows restless, is feverish at night, is thirsty, has a quick, hard pulse, and passes a small quantity of dark-colored urine. If care has been exercised, it will be found that the urine has been gradually diminishing for several days, and a slight puffiness about the face frequently announces the beginning of the trouble. Later the face becomes pale and puffy, while there may be edema of the feet and scrotum, and some vomiting. Under favorable treatment improvement may take place, large quantities of urine may be passed, and the child resume convalescence. The nephritic symptoms may, however, deepen until uremia appears, the pulse becoming slow, the temperature subnormal, and the tongue dry and brown. Vomiting is now a frequent occurrence; diarrhea is not unusual; nose-bleed and hemorrhages from the various mucous surfaces, and muscular twitchings may be noted, and most likely the end may be reached in a general convulsion. In all cases of nephritis great care must be taken to recognize heart-changes, and fatal results



are more frequent from cardiac failure than from the uremic convulsions. The constant effect of nephritis is to raise the blood-tension, and this continued tension in the blood-vessels is followed by dilatation of the heart. Another not unusual result is endocarditis or pericarditis, with possible embolism.

Sudden death frequently occurs during the course of nephritis. The child may be doing well, possibly sitting up in bed and playing with its toys, when an attack of dyspnea occurs; the face becomes livid, the pulse disappears, and death quickly takes place. Death in such cases is usually said to be due to edema of the lungs: the dilated heart, however, has been overlooked, and, while edema of the lungs is present, it is only secondary to the cardiac failure.

It is not unusual for a false membrane to form upon the larynx. This is not infrequently due to the streptococcus pyogenes, but the Klebs-Löffler bacillus is oftener found (Ranke found it in more than half of 92 cases). A bacterial examination should always be made early in the disease, and if the *diphtherial* nature of the infection has been determined the serum-treatment should be employed at once.

**Diagnosis.**—A typical form of scarlet fever offers few difficulties in diagnosis. The period of incubation is short in comparison with that of any of the other exanthemata, particularly variola, measles, and varicella. The vomiting, which is almost a constant factor in the early stage, associated with high fever, would also exclude the other eruptive diseases. The pulse in itself is most strongly diagnostic, being quick, hard, and wiry, striking the finger at the rate of 140 to 180 per minute; no other disease has a similar pulse. The early sore throat and the intense hyperemia of the whole mucous membrane, associated with severe constitutional symptoms, make it easy to differentiate from measles, varicella, and variola. The punctate erythematous lesions that appear during the stage of efflorescence of scarlet fever are not found in any of the other eruptive diseases. (*Vide* table on page 247.)

The differential diagnosis embraces the discrimination of those rashes that follow the use of certain drugs (quinin, belladonna, potassium bromid and iodid, chloral, etc.). The characteristic invasion-symptoms (vomiting, angina, etc.) of scarlatina are absent; also the high fever and frequent, hard pulse of the latter disease. Drug-rashes are seldom so vivid or diffuse as the eruption of scarlatina.

SCARLATINA.	ACUTE EXFOLIATING DERMATITIS.
Onset is sudden, with vomiting, angina, fever, and frequent, hard pulse.	Sudden, with fever only.
Eruption appears first on neck, face, and chest, soon becoming diffuse.	Appears first on trunk.
Duration, three or four days.	Duration, five or six days.
Desquamation begins after eruption has faded, often one week later.	Desquamation begins earlier, often before eruption has faded, and involves the hair and nails.
Ear and throat complications common.	Absent.
Nephritis is a common sequel.	Not so.
Relapses exceptional.	Relapses common.

The **prognosis** in regular, uncomplicated scarlet fever is in almost every case favorable, and, unless the treatment is unusually indifferent,



the patient will recover. Severe types, however, and especially malignant scarlatina, are very fatal. Complications arise that will most seriously endanger life.

The **treatment** of scarlet fever is that of the symptoms, together with an attempt at arresting the complications.

**Prophylaxis.**—The patient should be strictly quarantined in an upper room for at least eight weeks or until desquamation has been completed. A competent nurse should be put in charge, and, whether a member of the family or otherwise, she should wear a washable dress, and should not mingle with the family, except her clothing be changed or thoroughly disinfected. The room is to be stripped of all superfluous hangings and furniture. *Inunctions* are required as soon as desquamation commences, with a view to preventing the diffusion of the dried epidermal scales; and the best preparation for this purpose consists of cosmolin, menthol, and carbolic acid, ten grains each of the latter to one ounce of cosmolin, after the plan of J. Lewis Smith. Carbolyzed water, 1:40 (thoroughly shaken), may be used to sponge the surface and may be agreeably followed by cocoa-butter.

The disinfection of the physician himself, I am sorry to state, is frequently neglected. He should generate chlorin gas by the following simple method, and allow it to permeate his clothes thoroughly before going into other families: A dram of powdered potassium is placed in a saucer, and a small quantity of hydrochloric acid added. The dish is then placed on the floor, and the physician stands over the vapor chlorid as it arises until it penetrates all his clothing. This, with the free use of the whisk and thorough hand-washing, renders him non-contagious and safe in entering any home or sick-room. Perhaps a less disagreeable method is to have in the patient's house a linen duster or surgeon's apron that has been dipped in a bichlorid solution and allowed to dry. This is slipped over the clothing before entering the sick-room, and is removed after leaving.

In the room, if the case be a severe one involving the throat, I keep the gas or an alcohol lamp burning under a small dish of water, so that steam may be constantly generated. To the boiling water I frequently add carbolic acid or oil of eucalyptus; this saturates the room very pleasantly, and at the same time, I believe, limits the extent of the contagion.

**General Management.**—The sick-room should be large and well ventilated, and should be kept at a uniform temperature (68° to 70° F.—21.1° C.). A light flannel night-dress should be worn by the child, and the bed-clothing should be light as well. The *diet* should consist of milk, broths, egg-white, and fruit-juices, and after the temperature has declined soft diet may be allowed. A few days later the return to ordinary solid foods may be complete.

The evidences of heart-enfeeblement often arise and call for the judicious use of stimulants. It is to be pointed out that this class of agents is remarkably well borne in this affection, and hence may be freely administered. To a child of four years I give one dram (4.0) of brandy or whiskey every second hour, and often increase the dose as required. The preparations of ammonium, particularly the carbonate and the aromatic spirits, have also been warmly recommended as stim-

ulants in this affection. They should be administered in milk as the vehicle to prevent gastric irritation.

**Special Treatment.**—*Bathing* is recommended in scarlet fever by the best writers, yet often in such an indefinite and uncertain manner as to give the busy practitioner neither any encouragement to resort to it nor any guidance in the matter of technique and mode of procedure. In the classical work of Thomas Watson, now over fifty years old, he hints in his treatment of scarlet fever “that, if the heat on the surface be very great and distressing, he should not recommend the cold effusion, but cold or tepid sponging would be very refreshing and beneficial.” This sentiment finds its echo in most works on practical medicine at the present day. Unfortunately, the majority of medical practitioners do not give their instructions for the thorough sponging of their scarlet-fever cases, chiefly through fear of objections from the family.

The physician must quietly but firmly insist upon the patient being most thoroughly sponged three or four times daily, according to the severity of the individual case, using carbolized water (1:60), mercuric chlorid (1:8000), salt water, or alcohol and water, at a temperature of 70°–100° F. (21.1°–37.7° C.). Systematic bathing in this manner and inunctions as above described protect the body from certain disastrous complications and sequelæ. The ice-cap may be combined with cool spongings. In extreme cases, with marked nervous symptoms and high temperature, the cold pack, with cold affusions applied to the head and nape of the neck, may be cautiously employed, and a description of the method of giving a cold pack may be found under the treatment of Typhoid Fever. The gradually cooled bath may be substituted if open objection is made by the parents to the cold pack.

In regard to the use of *internal antipyretics*, I prefer phenacetin for older children, combined with quinin in capsules. Acetanilid is better for younger children, and I generally give one-half as many grains as there are years in the child's life. When medicine can be exhibited in the form of capsules, I always prefer to combine it with quinin or strychnin to overcome the tendency to depression. Phenacetin and acetanilid act successfully in controlling the nervous element, relieving headache and fever, promoting diaphoresis, and inducing refreshing sleep. Acetanilid is much more prompt in its action than phenacetin, but its effects are not so lasting. I therefore choose it for young children, and exhibit it in small doses in the form of a powder, and if the bowels are torpid I combine with the acetanilid small doses of calomel and soda. These agents are rarely required, and are not comparable in their good effects to hydrotherapy.

**Internal Antiseptics.**—Those remedies that are purely antiseptic, administered internally, have not given proof of their utility as yet. The sulfocarbolates of zinc and of sodium, on account of their breaking up in the system and liberating carbolic acid, cannot be used in a sufficiently large dose to meet with success. The syrup of phenic acid is used by many physicians, but their success does not as yet seem to warrant its being classed as an efficient remedy. Marmorek has used his antistreptococcus serum extensively, and, although it does not act as a specific, he claims that it prevents the serious complications and invariably renders the attack very mild.

The care of the *nose and throat*, and eventually of the *ears*, will require all the skill of the medical attendant, and by commencing early in the case to give careful and constant attention to these parts we may prevent much trouble and danger later on. The attendant should use a small atomizer filled with warm water containing a solution of sodium bicarbonate (gr. xv- $\bar{5}$ j—0.975-32.0). If decided inflammation should occur, a solution of hydrogen peroxid and cold water or glycerin (1 : 5) may be used, and then be followed by an oily preparation, such as liquid albolene containing menthol (a 5 per cent. solution).

If the patient cannot tolerate an atomizer, an application of the antiseptic oil directly to the posterior nasal spaces, by means of an aluminum applicator, may be made. Faithful attention to the removal of the secretion from the nose and throat will prevent accumulation, and thus prevent regurgitation up the Eustachian tube with its associated ear-troubles. In this way diphtheria can be prevented from gaining its full lodgement, and, if it gains ground at all, little trouble is experienced with this dreaded disease. For the appropriate treatment of this complication the reader is referred to the treatment of Diphtheria. If pain in the ear should indicate the extension of the trouble up the Eustachian tube, we must redouble our efforts, even though the desquamation within the Eustachian tube itself may be quite beyond the reach of our detergent wash.

The external auditory canal may also become blocked by desquamating epithelium, and this must be removed by gentle sponging. If the tension of the ear-drum becomes very great, it must be punctured. The crude method of dropping laudanum and sweet oil in the ear is to be condemned, as it serves as a nidus for a collection of dust and dirt, independent of the rapid accumulation of dead epidermis.

*Scarlatinal rheumatism* I have encountered in but a small proportion of cases, and then it was of a transient character, leaving no damaged heart-valves behind. I am inclined to attribute this fortunate result to the faithful use of daily bathing and inunctions, long continued and at least until after completion of desquamation.

The most constant complication of scarlet fever is *nephritis*. The specific poison of scarlet fever is peculiarly obnoxious to the kidneys, and is largely eliminated through them; and upon this fact hinges the scientific part of the treatment of this disease. The more active we render the skin the less likely will there be danger to the kidney. If the urine is examined throughout the whole course of the disease, we will find in the earlier stage that it grows less in quantity and becomes more laden with the waste of the body, at times being nearly suppressed by mechanical blocking of the uriniferous tubules. If now the skin is not invited to act to its fullest extent, we will soon find our patient reduced to a comatose state. Free bathing has the happy effect of vicariously eliminating the poison, and in this way it removes the undue pressure placed upon the kidneys. (For the treatment of nephritis the reader is referred to the discussion of acute Bright's disease under Diseases of the Kidneys.)



## MEASLES.

**Definition.**—An acute contagious disease, characterized by an initial coryza, general catarrhal symptoms, fever in the earlier stage, followed by a peculiar papular eruption on the face and body.

**Pathology.**—In uncomplicated measles we have no pathologic lesions. The only post-mortem changes found, as a rule, are those of catarrhal pneumonia and acute nephritis. All the internal organs are gorged with blood, and minute hemorrhages are found on their surfaces, while occasionally croupous pneumonia may be found involving a lobe or small portions of a lobe of the lung.

**Etiology.**—Measles occurs in epidemics, yet we have frequent sporadic cases in the larger cities. There is an epidemic prevalence in large centers of population every eighteen months or two years, but the different epidemics vary in their extent and fatality. It generally happens that when once the disease enters a home, street, or small court, scarcely any one escapes who has not been protected by a previous attack, those who suffer being for this reason, in nearly all cases, young children. The adults and older children may enjoy immunity in consequence of a prior attack, although this does not always follow. The susceptibility to measles in children, however, is very great. A most notable example of this was the epidemic that occurred in the Fiji Islands in 1875, and raged for four months, 40,000 of the natives dying out of a population of 150,000 (Corney, quoted by Callir). Biedert<sup>1</sup> found that only 14 per cent. of unprotected children escaped. In the Faroe Islands, under similar conditions, only 1 per cent. escaped (Madsen, Pannum). There is the same experience in schools and hospitals: when a case incubating the disease is admitted, the whole unprotected junior population is attacked. The epidemics occur mostly in the fall and winter, yet the season seems to have but little influence.

**Bacteriology.**—Micrococci, especially *streptococci*, have often been found in the secretions of the respiratory tract, but they have not been proved to be specific, as they are not always found and cultures from them do not cause the disease.

Canon and Pielicki<sup>2</sup> found in the blood of 14 cases, as well as often in the sputum and nasal and conjunctival secretions, a special bacillus of irregular size, which colored irregularly with methylene-blue. It was decolorized by Gram's method, did not grow on solid media, but did occasionally on bouillon.

Czajkamski<sup>3</sup> described motile bacilli, 2.5 to 5 micromillimeters in length, which did not color by Gram's method. They could not be cultivated on gelatin or agar, but grew on glycerin-agar, bouillon, and blood-serum, and killed mice by producing septicemia. Neither of these observations has been advanced to any firmer position in the etiology of the disease.

One attack of measles does not seem always to exhaust the soil, as in the other exanthemata: one, two, and in several cases families of children, including the parents, have had four attacks in successive years.

<sup>1</sup> *Jahrbuch. für Kinderheilkunde*, vol. xxiv. p. 94.

<sup>2</sup> *Berliner klinische Wochenschrift*, 1892, S. 377.

<sup>3</sup> *Centralblatt für Bacteriologie*, vol. xviii. Nos. 17 and 18.

**Clinical History.**—The period of incubation is from seven to fourteen days, and in inoculated cases from seven to ten days.

**Catarrhal Stage.**—The early symptoms are those of a cold with some fever. The child has marked coryza, watery eyes, sneezes, and has a dry, croupy cough. Frequently the symptoms are those of a catarrhal laryngitis and bronchitis, the fauces and tonsils being hyperemic, with abundant secretion; and, in addition, an examination of the eyelids reveals a conjunctivitis. The patient may be acutely ill, the temperature rising several degrees in the evening, and falling slightly in the morning; the fever continues high until the rash is fully developed. The rash, consisting of one or more distinct papules, may be seen on the hard palate fully twenty-four hours before it appears on the face.

**The eruptive stage** is very characteristic, and usually makes its appearance at the end of the fourth day. The neck, face, forehead, and trunk receive the eruption in the order of mention. The whole physiognomy of the child is so characteristically altered that a well-marked case may be diagnosticated at a glance. The face is flushed; the eyes are red and watery; a short, dry cough, frequently metallic in ring, is present; and the nose and cheeks are covered with crops of dusky-red papules surrounded by a zone of erythema which sharply contrasts with the normal skin between the patches. The rash on the face is both discrete and confluent, or may be arranged at times in small crescents, and in the course of a day or two the whole trunk is invaded, but in a slighter degree. By the fifth, and seldom the sixth day, the eruption has reached its height, and commences to fade, first on the face and neck, then on the body and limbs, followed by a fine desquamation. By the seventh or eighth day the rash is nearly gone, leaving a blue,

mottled stain over the body. The temperature, which has reached 103° F. (39.4° C.) or even 105° F. (40.5° C.), falls when the rash is fully established—*i. e.* on the fifth or sixth day—while the headache, the severe bronchial cough, and the general features subside with the fever. If the temperature continues high after the rash is out, we may look for some complication, such as severe bronchitis, pneumonia, or acute nephritis (*vide* Fig. 23).

**Complications.**—In some epidemics the character of the disease is very severe, being marked by high fever (105°–

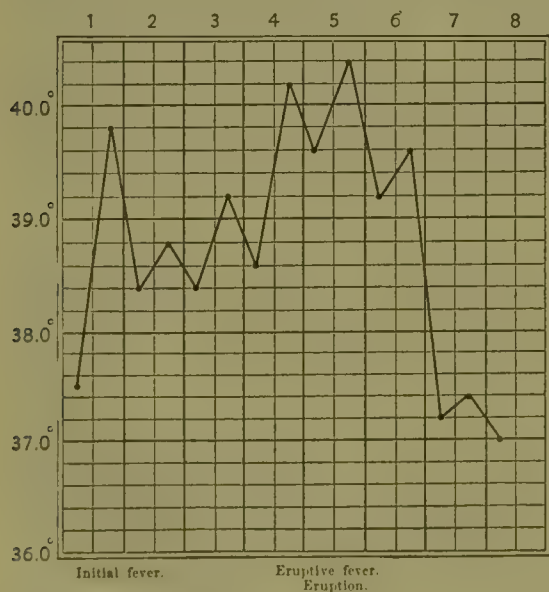


FIG. 23.—Temperature-curve of a case of measles.

106° F.—41.1° C.). a dry, brown tongue, delirium and convulsions, and feeble heart-action, due to the intense hyperemia of all internal organs—lungs, brain, kidneys, etc. I have observed cases in which the eruption was petechial.

The main complications are presented by the lungs. The accompa-

nying bronchitis manifests a strong tendency to extend to the bronchioles, with resulting *broncho-pneumonia*. The extent and seriousness of this complication are largely dependent upon the degree of the previous debility. *Lobar pneumonia* is rarely met with.

*Catarrhal* or *membranous laryngitis* is frequent in the pre-eruptive stage or as a sequela. Quite rarely edema of the glottis occurs. *Ophthalmia* may occur in anemic and strumous children if strict eye-toilet is not enforced. *Glandular involvement* may take place in the cervical glands. *Otitis* is frequent during desquamation, suppuration taking place in the middle ear and the membrane being perforated. This may be avoided, however, by cleansing the post-nasal spaces frequently during desquamation. *Cancerum oris* and *noma pudendi* may also appear as complications of the disease. *Diarrhea* is frequent at the end of the eruptive period and as a sequela.

The health of the child often remains impaired for a long time after an attack of the measles: it is at this period that whooping-cough, diphtheria, nephritis, and, later on, *acute tuberculosis*, may arise. Tuberculosis very frequently gains entrance into the system from the existence of enlarged and cheesy bronchial and mediastinal glands. Nervous sequelæ occur, but are very rare. They usually take the form of paralysis (hemiplegia, paraplegia, etc.).

**Diagnosis.**—The disease is the most variable of all the exanthemata. Epidemics may be characterized by irregular forms of the disease, and the diagnosis of sporadic cases is often very difficult. We cannot recognize it by its dermal lesions, but by the prodromal symptoms, by the fall of temperature after the eruption is well out (differing here from scarlet fever), and by the character of the pulse, tongue, and desquamation. A feverish period of four days, associated without catarrhal symptoms of the eyes, nose, and upper air-passages, a few papules on the hard palate, followed within twenty-four hours by a papular efflorescence on the face, will differentiate the disease from variola, varicella, scarlet fever, and rubella.

The accompanying table, from Rotch, gives the diagnosis between the eruptive diseases at a glance:

	MEASLES.	VARIOLA.	VARICELLA.	SCARLET FEVER.	RUBELLA.
Incubation . . .	10 days.	12 days.	17 days.	4 days.	21 days.
Prodromata . . .	3 days.	3 days.	A few hours.	2 days.	A few hours.
Efflorescence . . .	Papules.	Macules. Papules. Vesicles. Pustules.	Vesicles.	Erythema.	Papules.
Desquamation . .	Purpuraceous.	Large crusts.	Small crusts.	Lamellar.	
Complications and sequelæ . . .	Eye and lung.	Larynx. Lungs.		Kidney, ear, and heart.	

The mortality differs according to the surroundings of the patient. In healthy children under favorable environment the mortality is practically *nil*, while in tuberculous and wasted children it is very large, this being especially due to complications and sequelæ. Infants may be born with the rash on them.<sup>1</sup>

<sup>1</sup> *Hem. Med. Chronicle*, May, 1890; *Brit. Med. Journal*, vol. i. p. 612, 1890.



**Treatment.**—We are unable to shorten the disease, though it is self-limited; nor is there any means of producing immunity from the disorder. The treatment is necessarily symptomatic; hence our efforts should be directed to protecting the various organs that are most likely to become involved by complications, remembering at the same time that the nose, ears, eyes, and throat are involved during the feverish stage, and that the skin is in a very susceptible condition.

The patient should be placed in a large dark, well-ventilated room, with a uniform temperature between 68° and 70° F. (21.1° C.). He should remain in bed until the temperature has been normal for one week, and until the efflorescence has nearly faded and the desquamation is almost complete. The diet during the period of fever should be milk, bread, and light soups. Near the end of desquamation, if all symptoms are favorable, a more generous dietary may be allowed.

The bronchial cough, which may be very troublesome during the first few days, can be readily relieved by some simple expectorant mixture, as—

R <sub>x</sub> . Potassii citrat.,	℥ss (16.0);
Succi limonis,	℥j (32.0);
Tr. opii camph.,	℥ij (8.0);
Syr. ipecac.,	℥ij (8.0);
Syr. tolu.,	q. s. ad ℥ij (64.0).—M.

Sig. ℥ss-℥j every two or three hours, according to the age and condition of the patient.

This will serve as a fever mixture as well as an expectorant.

For the coryza I have found that atomizing the nares with some oily vehicle (oleum petrolatum album, etc.) is advantageous.

The skin is in a state of great irritation, and from the very commencement of the disease until the end of desquamation a daily warm bath (95° to 100° F.—35° to 37.7° C.) should be given the patient. The body should be carefully dried and cocoa-butter then thoroughly rubbed over the entire surface. The child should live in an equable temperature for at least three weeks, and longer if desquamation has not then ceased. For months he should be protected from sudden atmospheric changes in order to avoid general respiratory troubles. If he be predisposed to tuberculosis, cod-liver oil and creasote should be prescribed for a period of two months or more.

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## RUBELLA.

(*Rötheln*; *Rubeola Notha*; *German Measles*; *French Measles*.)

**Definition.**—An acute contagious disease. It has no prodromal stage, and is characterized by slight fever, coryza, and an efflorescence upon the skin.

**Etiology.**—Rubella was not distinguished from measles and scarlet

fever until about the middle of the eighteenth century. Since then considerable controversy has arisen at different times as to its nature, the theory being at one time strongly advanced that it was a combination of these two diseases, as many of the milder cases have symptoms common to both. That there is a difference, however, in the character and course of these diseases has been proved beyond doubt to careful observers by the facts that rubella occurs independently of either measles or scarlet fever; that contagion from this disease produces a similar disease; that one attack affords immunity to subsequent seizures (although those who have had other eruptive diseases are as liable as those that have not to contract this disease during an epidemic); and that its onset and clinical course are characteristic.

Rubella is contagious, and may occur epidemically or sporadically. It is like measles and the other exanthemata in its being of undoubted microbic origin, although, as is the case with them, the specific organism has not been isolated. When that is accomplished the diagnosis will be more easy and certain.

In hospitals or where persons are crowded and living under unhygienic circumstances the disease is very contagious and the epidemic will be quite general; but in family practice it is but slightly so, and the epidemics are limited, often being confined to a single household and attacking perhaps but one or two of the family. As stated by Edwards, it is spread by the cutaneous exhalations, breath, fomites, and clothing, and is probably contagious from incubation until far into convalescence.

**Clinical History.**—The incubation stage lasts from ten to twelve days, though this period may vary and the disease appear three or four days after exposure. On the other hand, cases have been reported in which it was as long as three weeks. As a rule, the period of incubation is longer perhaps than in measles. The **stage of invasion** covers from one to three days, but in mild cases the rash is very often the first indication we have that the child has developed an infectious disease.

For a period of a few days before the rash appears there will be noticed chilliness, pains in different parts of the body, a dull, heavy feeling, perhaps feverishness, accompanied by sore throat, enlarged tonsils, coryza, and suffusion of the eyes, constriction over the chest, and a dry cough and bronchitis. Enlargement and induration of the cervical and other lymphatic glands, together with the sore throat, are common symptoms.

Just before, or with the appearance of, the rash there is a rise in temperature to 99° or 100° F. (37.7° C.), or in severe cases as high as 103° F. (39.4° C.) or more. Again, the invasion symptoms may be absent or so mild as to escape notice, and the first sign of infection be the appearance of a rash which first shows itself on the face and extends downward over the body. In some cases it does not follow the regular course, and is confined to one part of the body, and cases have been reported in which it only appeared on the roof of the mouth or on the tonsils. In other cases every part of the body, including the palms of the hands and the soles of the feet, may be covered.

The eruption consists of papules, is multiform, confluent, and of a pale or rosy-red color. The patches do not assume any regular shape

or form, and the skin between them may become hyperemic and cause itching. The rash reaches its height on different parts of the body in succession, fading in one part while appearing in another. Its duration is from two to five days, and possibly longer in some cases.

A slight desquamation usually occurs, and a slight pigmentation of brownish color after the rash fades is frequently noticed, disappearing after a few days. The temperature-curve is variable, but as a rule it remains between 100° F. (37.7° C.) and 102° F. (38.8° C.) while the eruption is present. As mentioned above, sore throat is nearly always present, with enlarged tonsils, a dry cough, and bronchitis. The glandular enlargement will also continue with the rash, and in severe cases the axillary and inguinal glands may become involved. The pulse varies with the temperature and respiration. Vomiting has been noticed as occurring during the eruption in severe cases.

After a period varying from three days to a week, with the disappearance of the rash, convalescence begins and the child rapidly regains its former health, and the whole course of the disease may be so mild that the patient cannot be persuaded to remain in bed.

**Complications.**—The most common are affections of the *respiratory tract* (pneumonia or severe bronchitis), and in some cases we have a *gastro-intestinal catarrh* of a troublesome character. *Diphtheria* or other contagious diseases may occur. A relapse is not uncommon, and may be as severe as the initial attack.

**Diagnosis.**—Rubella may be distinguished from *measles* by its less severe onset and course, by the lighter color and more diffuse character of its rash, and by the irregular shape which the patches assume. The presence or absence of an epidemic is an important factor in the diagnosis, and in cases occurring when there is no epidemic the diagnosis between this disease and measles of a mild type is difficult if not altogether impossible.

From a well-marked case of *scarlatina* the diagnosis offers no difficulty. The absence of its initial vomiting, the strawberry tongue, the character of the rash (which in scarlet fever is erythematous), and the shorter duration and milder course of rubella, all help to render the diagnosis easy.

RUBELLA.	ERYTHEMA.	URTICARIA.
Occurs first on the face.	On the hands and feet.	In wheals on arms and legs.
Marked coryza present.	No coryza.	No coryza.
At first no itching.	Burning pain.	Intense itching.
Contagious.	Not contagious.	Not contagious.
Microbic origin.	Reflex origin.	Gastric origin.

The **prognosis** in uncomplicated cases is invariably good, but when the surroundings are unhygienic, or in cases in which the child has been delicate previously, it is more serious. Complications, especially pneumonia or diphtheria, may prove fatal, and in some cases the mortality reported has been as high as 9 per cent.

**Treatment.**—The treatment is simple and principally symptomatic. A mild cough-mixture, such as is recommended in measles for the bronchitis, nutritious but easily digested food, and medicine to regulate the bowels when necessary, fulfil all the indications for internal medication.



As in measles, cool sponging should be resorted to before and during the rash; and, when the fever is high, a cool tub-bath, where practicable, will be found to reduce the temperature, quiet the patient, and hasten the appearance of the eruption. During convalescence, if the child does not rapidly regain his appetite and strength, tonics, such as tincture of nux vomica and syrup of hydriodic acid, are indicated.

The complications are to be treated as they arise, but the sponging should not be discontinued until the temperature reaches its normal level.

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## WHOOPING-COUGH.

(*Pertussis*; *Tussis Convulsiva*; *Keuchhusten*.)

**Definition.**—Whooping-cough is a highly contagious disease which is characterized by a catarrhal inflammation of the respiratory tract, associated with a peculiar spasmodic cough, ending in a whooping inspiration.

**Pathology.**—There is no lesion that can be considered characteristic of whooping-cough, and there is no distinct causal lesion around which all the symptoms and complicating lesions are grouped. In the beginning there is catarrh of the naso-pharynx, and this may be the only lesion coincident with the development of the characteristic cough. In advancing cases this naso-pharyngeal catarrh becomes generalized by extension to the lachrymal ducts, the conjunctivæ, the Eustachian tube and the middle ear, to the glottis, trachea, large and small bronchi, and the air-vesicles. The more decided pulmonary lesions—emphysema, pulmonary collapse, pulmonary congestion and edema, and broncho-pneumonia—are advanced pathologic conditions accompanying the later stages or more intense forms of the disease (W. W. Johnston).

The *post-mortem* table does not give us much information as to the pathology except as to the sequences of the disease. In the early stages swelling and redness of the respiratory and digestive tracts will be found, together with a large quantity of viscid mucus.

**Etiology.**—The disease occurs in *epidemics*, yet occasionally may appear *sporadically*. *Pertussis* seems to have a tendency to occur in epidemics every two years, although in large cities the disease is generally endemic. There is no doubt that it should be classed with the specific diseases, yet for a long time, like mumps, it hovered between the specific and the catarrhal diseases for a time. *Pertussis* is directly contagious, though scarcely so in houses and school-rooms unless it be for those of a specially susceptible nature. It is possible, however, for the disease to be propagated in schools, though not to the same extent as measles and scarlet fever. It seems that a more decided and prolonged personal contact must be made, as with members of a family, to ensure transmission. One attack practically protects the child, yet exceptions to this rule may be found. The influence of the *seasons* does not seem to have any effect, though perhaps fall and spring are the more frequent periods; the station in life, whether hygienic or unhygienic, does

not modify the disease. *Bad ventilation*, however, may propagate the disorder, and cause additional cases by favoring the increase of germs in the immediate surroundings. The *previous condition of health*, especially of the respiratory mucous membrane, seems to possess some predisposing influence, weak, delicate children with an irritable digestive tube associated with a catarrhal state, more readily contracting whooping-cough than those in robust health.

There seems to be an intimate association between whooping-cough and *measles*, and it is a well-recognized fact that an epidemic of measles will be followed by whooping-cough in the same sufferers. This is possibly due to the sensitive condition of the mucous membrane left by the measles, which is so favorable to the lodgement of the germs of pertussis; and the association of the two diseases must be more than accidental. There exists a certain individual susceptibility to whooping-cough, as well as to other infectious diseases, and yet many children never contract them, though frequently exposed.

*Age* exercises some influence on the development of whooping-cough, most cases occurring before the tenth year; after this time the frequency of the disease rapidly diminishes. West states that one-half of all cases develop under three years, but he must have based his knowledge upon an experience in hospitals and children's homes, as the experience of others does not sustain his statement. The disease occurs in adults but rarely, this being due partly to the fact that so many have suffered from it while young, and partly because of a lessening of the susceptibility with advancing years. It occurs frequently before the first year, and when it does it is the most fatal of all the diseases of childhood (Goodhart).

The *sexes* are about equally divided as regards susceptibility; many writers, however, seem to think that girls are most liable. Ofttimes one close exposure in a susceptible child is sufficient to ensure an attack. The germs seem to be located at first in the secretions of the respiratory tract, and are thus disseminated through the air, the disease being most highly contagious, therefore, during the paroxysms of coughing. Goodhart reports a case in which a third party was the medium in conveying the disease from one child to another, thus suggesting a possibility of the contagion being ponderable.

The highway of the contagion of whooping-cough into the system is evidently through the respiratory tract, though this fact has not yet been definitely settled. Published cases of pertussis in the new-born would even seem to make its transmission possible through the fetal circulation, yet the reports are neither numerous nor satisfactory, and cannot be depended upon.

**Nature and Bacteriology.**—The true nature of whooping-cough has been thoroughly discussed, but is not, as yet, fully settled. Many writers claim it to be a simple bronchitis due to "cold" associated with a certain nervous habit or mimicry. The cough is started by the bronchial irritant, and soon tends to become a habit, thus returning again and again, until it dies out in the oblivion engendered by more healthy and regulated discharges of nervous energy (Goodhart). This theory fails to account for the nervous element and the decided paroxysmal character of the cough. It has been held that the disease is a lesion of

either the pneumogastric, phrenic, sympathetic, or recurrent laryngeal nerves, or perhaps even of the medulla. If this ground is valid, it is simply a neurosis. Eustace Smith says it is caused by the pressure of the enlarged tracheal and bronchial glands upon the terminal filaments of the pneumogastric nerve. Whatever the direct cause, the highly contagious character of whooping-cough, its appearance in epidemics, its incubating period, and the possible immunity from subsequent attacks seem to prove beyond argument that it should be classed among purely infectious diseases.

While this is generally accepted as true by the most recent investigators, it is not a new idea, and science only repeats herself in this instance. Linnæus (to quote Dr. J. P. C. Griffith in the *American Text-book of Diseases of Children*) attributes pertussis to the presence in the nose of the larvæ of insects. Poulet discovered bacteria in the expired air of patients suffering with the disease. Letzerich found a micrococcus in the sputum which he believed to be the specific germ, and claimed to have been able to produce the disease in animals by introducing the secretion into the trachea. Deichler-Kurlow claimed that there was always present in the sputum an organism of the nature of a protozoon which possessed ameboid motion. And, while other investigators have repeatedly described various organisms as existing upon the respiratory mucous membrane, the researches of Afanassieff in 1887 have attracted the most attention. This observer isolated a short bacillus, which he named the *bacillus tussis convulsivæ*, and of which he was able to obtain pure cultures upon various media. Animals inoculated upon the respiratory mucous membrane with these cultures exhibited some of the symptoms of the disease and developed catarrhal conditions of the respiratory tract, with a tendency to broncho-pneumonia. These observations have been confirmed by others, and a toxin has also been reported as present in the urine of patients suffering with pertussis which is identical with that produced by Afanassieff's bacillus. Even though it be admitted as most probable that some micro-organism is the cause of the malady, it is by no means clear how the symptoms are produced or where the principal seat of the infection arises. Some writers have claimed that the trigeminal nerve is in a sensitive state, and that it is the irritation of its terminal filaments by the infectious catarrhal process on the nasal mucous membrane which brings on the paroxysms by a reflex action. The careful investigations of Myer-Huni and of von Heroff, however, indicate that the catarrhal inflammation is most pronounced in the mucous membrane of the nose, larynx, and trachea down to the bifurcation, but especially so on the posterior wall of the larynx in the interarytenoid region, the so-called "cough region." Kuoloff believes that the parasite of whooping-cough is a specific micro-organism, a protozoon, and has found uniformly in the fresh sputa of patients ameboid organisms with spheric spores characterized by concentric laminations.<sup>1</sup> Undoubtedly we have in whooping-cough an infectious catarrhal process which affects the mucous membrane controlled by the superior laryngeal nerve, and the value in many cases of purely local treatment indicates that the abode of the germs is in this region, whence the poisonous products of their growth are absorbed.

<sup>1</sup> *Medical News*, Nov. 9, 1896.



The nature of the "whoop" has been frequently discussed to show the nervous origin of the disease, yet the infantile larynx is capable of responding to purely neutral stimuli owing to the flexible nature of the young cartilage. If we carry a young sleeping child from a warm room out in the cool air, the same characteristic whoop may be produced, showing that this reasoning cannot be depended upon.

**Clinical History.**—The period of **incubation** varies from four to fourteen days according to the extent of catarrhal trouble in the child existing at the time. Goodhart gives several authenticated cases in which the incubation ended on the eighth day. In the beginning the symptoms are those of a slight bronchial cough, which has a tendency to be more pronounced during the night. After a few days the cough assumes an influenzal character, and at the same time it gradually grows metallic in ring and shows a laryngeal type. There is some fever present. This **catarrhal** or **feverish** stage lasts for a week or more, when it is followed by the **paroxysmal stage**, and these stages are divisions of the symptoms worthy of recognition, as the treatment in the first is not applicable to the second. Many authorities speak of a third stage as one of **decline**, which does not sharply occur, but includes the sequence of the disease. The **catarrhal stage** lasts about one week or ten days, during which the child is ill at ease, is feverish, and has a hoarse, dry cough. The symptoms may either be entirely laryngeal at first or bronchial, with a loss of appetite and broken rest at night. Auscultation at this time will reveal a few moist or dry râles in the larger bronchial tubes, but there is very little secretion. The cough seems to be out of proportion to the physical signs. As the catarrhal stage proceeds the cough commences to indicate its character by becoming more noisy, increasing especially at night. The physiognomy of the child commences to change, the face is swollen, the eyes suffused and watery, the under lids swollen and pink in color. This is one of the most decisive indications of the trouble, and may be recognized by a careful observer a few days before the "whoop" begins which stamps the disease and ushers in the second stage. The commencement of the **paroxysmal stage** is quite different from the easy and more constant coughing of the first stage. If the child is in bed, the onset of a paroxysm is usually quite sudden, but if he is up and playing, there is a period of restlessness, a premonition of the coming storm similar to the aura in epilepsy, and the child may even have time to run to his mother or nurse before the paroxysm comes on. Usually the paroxysms are induced by a quick inspiration, as during drinking, eating, or crying. The first (expiratory) part is short, and followed by a short whoop; this is very quickly followed by a long series of short expiratory efforts and a second and longer whoop, when the paroxysm may cease. In some cases a third and a fourth may quickly follow, until the child is quite exhausted. The paroxysms, whether short or long, generally terminate with vomiting or eructation of a quantity of stringy mucus. Food is ejected, and in most cases a little blood is mixed with the vomited mucus.

At this stage of the disease, if at all severe, the countenance of the child is characteristic, and so much so that a mistake is no longer possible: the features are swollen, puffy, and dusky in color; the eyes are injected, the lids swollen and pink; the skin livid, due to a minute

ecchymosis of the smaller capillaries. In many cases there will be extravasation of blood beneath the conjunctiva, due to the violence of the congestive cough. If the chest be examined at this stage, it will tell but little, provided we have no broncho-pneumonia, though a few moist râles may be found scattered through the larger tubes.

The spasmodic stage of whooping-cough has no set duration and varies frequently in intensity. In severe cases it may consist of twenty to forty paroxysms during the twenty-four hours. Some spasmodic coughs are not accompanied by a whoop, and the absence of this sign may be noted in very young children, as well as in those that are very ill with broncho-pneumonia. Some children vomit after a coughing spell without the whoop.

It is frequently observed that long after the spasmodic spell has come to an end the paroxysms return again and again, perhaps years afterward, with almost characteristic features, evidently acting under the stimulus of some perfectly neutral catarrh.

**Complications.**—In severe cases the complications are likely to be numerous.

*Epistaxis* often occurs in children; *hemoptysis* when vomiting is frequent; *ulceration* of the frenum linguae in violent coughing; *convulsions* in vigorous children; and *broncho-pneumonia*, *pleurisy*, *pericarditis*, *laryngitis*, and *hernia* in severe, prolonged coughing. Convulsions and broncho-pneumonia are alarming; in young children a *profound stupor* takes the place of the convulsions, and the latter then become of graver significance.

**Sequelæ.**—*Acute nephritis* frequently occurs, and is as severe as that found in scarlet fever, although the condition has not received the recognition it should from the authorities. In a series of over 200 cases I have found the kidneys affected in 20 per cent. *Emaciation* is a very important sequence of pertussis. All the viscera are liable to fatty degeneration, and nutritional changes open the door to cheesy, glandular alterations, and eventually to a secondary tuberculosis. Atelectasis, by curtailing lung-space, frequently brings about a general collapse, and this condition very frequently explains the flattened chest found in young adults. Emaciation may also be due to *mucous disease*, a chronic gastro-intestinal catarrh of long standing.

**Prognosis.**—Associated with its complications, pertussis is a very fatal disease, especially in children under two years of age. Dolan regards it as third in rank among the fatal diseases of England, where the death-rate per million is five thousand annually. The deaths occur chiefly among children of the poor and in bottle-fed infants.

Goodhart regards whooping-cough as the most fatal of all the diseases in children under one year of age. He places the mortality as high as 12 per cent., and thinks that this is not too high; his statement, however, is hardly warranted, as he includes the deaths from the many sequelæ which we cannot estimate. Ashby and Wright place the mortality at 7.6 per cent.

**Diagnosis.**—Young infants usually do not “whoop,” but cough spasmodically. Children with *pleurisy* or *pneumonia* do not whoop, yet we diagnose whooping-cough by the preceding catarrhal fever. From *influenza* in its early stages it is most difficult to differentiate the

affection. The pink under eyelid has to me been the most certain sign. When the whoop appears and during the existence of an epidemic, however, the diagnosis may be rendered certain.

The diagnostic point prior to the whooping stage, enunciated by Eustace Smith (viz. "If a child be made to bend back the head, so that his face becomes almost horizontal, and the eyes look straight upward at the ceiling above, a venous hum, varying in intensity according to the size and position of the diseased glands, is heard with the stethoscope placed upon the upper bone of the sternum. As the chin is now slowly depressed the hum becomes less loudly audible, and ceases shortly before the head reaches its ordinary position") has not been very satisfactory. It is true that we do recognize the hum caused by the enlarged bronchial gland, but it occurs long after other symptoms are manifest, and thus its importance is much lessened.

I have for several years been able to place considerable value on the peculiar puffiness of the mucous membrane of the eyes and the swollen or edematous condition of the whole face and almost dusky color. This condition may exist for days before the catarrhal symptoms have extended throughout the respiratory mucous membrane. The cough at this stage may not be at all suggestive: it may be, in fact, purely bronchial.

This symptom of fulness about the eyes, which is quite as constant as in measles, would in fact suggest that disease, and must be differentiated from it. As we are able to diagnose measles by its appearance first on the hard palate, so I contend we may diagnose whooping-cough in its earliest stage by the characteristic swollen condition of the eyes and face. I insist upon this factor as of the greatest importance, as its recognition will enable us to institute specific treatment early, when the disease is yet local and may be brought more speedily under control.

**Treatment.**—The gravity of pertussis is scarcely appreciated either by the general physician or the public, and there is more criminal neglect in connection with whooping-cough than with any other disease. Medicinal treatment is exceedingly unsatisfactory, although the therapeutic measures are boundless: just as in phthisis and diphtheria, they cover the whole domain of the Pharmacopeia, and we have as yet found no specific. The remedies most in use are the antispasmodics and the germicides.

Whooping-cough has a striking parallel in diphtheria, in that it has in its early stages a local manifestation in its strong tendency to fasten itself upon the throat. How long this period exists we know to a certainty no more than we know just how long diphtheria is purely a local throat-poison; yet there is undoubtedly a period in whooping-cough, as there is in diphtheria, long or short, in which the virus—if it could be recognized—could be destroyed and the disease terminated. To abort cases thus within two weeks is not unusual, and this explains the number of reported cures made by germicidal remedies.

I have notes of 2 recent cases in which the characteristic whoop commenced at once with the general catarrhal symptoms, and was cut short by a hydrogen-peroxid gargle. I think these 2 cases illustrate very clearly the fact that the germs of the disease will locate on the mucous



membrane of the respiratory passages and bring about a nerve-discharge which ends in the characteristic whoop. In my treatment of this disease I find the greatest necessity of recognizing the nature of the trouble early in the catarrhal stage. If I can satisfy myself that I am dealing with a case of early pertussis, my methods of procedure are much different from what they would be if the case were well advanced. We must remember that the two stages are not sharply defined, and that many cases entirely lack the catarrhal stage, just as there are many cases that do not whoop.

Pertussis, as we all know, is a notoriously unsatisfactory disease to manage, and if we put our whole confidence on a single remedy, we are likely to meet with keen disappointment. The drugs I have found most efficient in the catarrhal stage have been hydrogen peroxid in sterilizing the naso-pharynx, and asafetida, occasionally used for the paroxysms. Belladonna is to a high degree beneficial in young children. I push this drug until I get the full toxic effects, when I am generally rewarded with a diminution of the suggestive characteristics of the cough.

To be more explicit, I will detail the methods of procedure in a family in which I have instituted my plan of thorough treatment: A child of four years attending kindergarten was brought to me with a suspicious cough. The history was given of an exposure of over two weeks prior. The child had coughed for a few days, more at night than in the daytime; was feverish during the evenings; showed slightly swollen eyelids, thus suggesting the nature of the impending trouble. I ordered hydrogen peroxid and pure glycerin in equal parts, which were well diluted and thoroughly sprayed through the naso-pharynx every four hours. The diet was light and digestible; out-door life was encouraged, except on windy days. All excitement was avoided, so as to avoid the precipitation of any additional paroxysms. At night the child was placed in a large, well-ventilated room, and over its cot was erected a mosquito netting, so as to prevent any unusual draught—a procedure which I have found highly beneficial, as it materially lessens the number of the nocturnal paroxysms. When the cough was fully established and was accompanied by eructations of stringy mucus, I commenced the exhibition of the mixture of asafetida  $\frac{1}{2}$  dram (2.0) every two hours. The record of the paroxysmal stage was as follows: The first week averaged six coughing spells per day; the second week averaged ten per day; the third week, four paroxysms; and the fourth and fifth weeks averaged about two paroxysms during the twenty-four hours. When the younger brother, but eight weeks old, commenced to show evidences of the disease, I first used hydrogen peroxid as in the older brother, and immediately followed it with asafetida. This case continued scarcely four weeks when all symptoms subsided.

Mistura asafetida, however, is at times disappointing, even in younger children. My second choice is the tincture of belladonna, exhibited in doses of one drop for every month of the child's life, the doses being rapidly increased until toxic effects are reached. Then I gradually increase the amount as tolerance of the drug seems to be established. In very young children I have obtained good results from the use of a freshly-prepared belladonna plaster placed between the scapulæ, and the physiologic action of the drug seems thus to be more constantly

maintained. The plaster may be changed at the end of one week. In a number of very troublesome cases in young children I have gained a decided advantage by an application of a 2 per cent. cocain solution directly to the naso-pharynx. This treatment, however, does not preclude the use of hydrogen peroxid, which should be continued throughout the catarrhal stage.

Bromoform was resorted to in fully 20 per cent. of my cases, and was a keen disappointment; it seemed merely to stupefy the patient and did not apparently shorten the progress of the disease. The coal-tar products, pushed to the toxic limit, modified the disease but slightly. Belladonna and antipyrin in combination gave better results than either alone. Quinin, chloral, creasote, carbolic acid, I found to be of little practical use, owing largely to difficulty in administration. This outline of the drug-treatment in whooping-cough has reference solely to the catarrhal and paroxysmal stages of the disease. As important adjuncts to the management of the disorder careful hygiene must be enforced, and a diet of the simplest character and a uniformly quiet life maintained. Throughout the whole course of the disease out-door life, as far as possible, should be encouraged, and if convenient a sojourn at the sea-shore will shorten the progress of the trouble and limit to a great extent the number of sequelæ.

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## PAROTITIS.

(*Mumps; Parotiditis; Epidemic Parotitis.*)

**Definition.**—An acute contagious disease, characterized by an inflammation and swelling of the parotid gland, and occasionally by an involvement of the salivary glands, the testicles, and in the female the mammæ.

**Pathology.**—Opportunities for post-mortem examinations are rare, leaving in some doubt the pathologic course of the disease; but it probably begins as a catarrhal inflammation of the ducts, involving the periglandular connective tissue. The inflammation is seldom severe enough or of such a nature as to produce suppuration.

**Etiology.**—Mumps is undoubtedly a constitutional or blood-disease with local manifestations. "It is a question," Goodhart says, "with mumps whether this disease shall be placed with the specific diseases or with those affecting the parts or organs with which the symptoms more particularly concern themselves."

The disease is no doubt of microbic origin, but the specific organism has not yet been isolated, and, while there has been some reason to believe that it is a bacillus, this has not been proved and is still doubtful. It is highly contagious, and at times, usually during the spring and autumn, becomes epidemic. It is communicated principally by the breath and exhalations, the greatest source of contagion being the salivary secretions. It may, however, be carried by a third person or by fomites, and is most liable to be communicated during the begin-

ning of the attack, although the contagiousness continues until after the subsidence of the febrile symptoms. It occurs mostly among children and young adults, infants and old persons being rarely affected, while males are more liable than females. One attack usually gives immunity from a second attack in the same gland.

**Clinical History.**—The average period of incubation is fourteen days, but it may develop as early as ten or as late as twenty days after exposure. The invasion is marked by languor and a temperature from  $101^{\circ}$  to  $103^{\circ}$  F. ( $38.3^{\circ}$ – $39.4^{\circ}$  C.), with possible headache and vomiting; the patient complains of pain at the angle of the jaw, and this is greatly increased if an acid (such as vinegar) is swallowed. With these symptoms is noticed a pyriform swelling of the parotid glands, the one on the left side usually appearing first, and the other one soon following. Occasionally cases are seen in which but one gland is involved, or the swelling may begin in both at the same time. This increases gradually until some time between the third and sixth days, involving the other salivary glands and causing marked disfigurement; the swelling fills the depression beneath the ear and extends to the cheek and neck, the most prominent part being just below and pressing outward the lobe of the ear. The salivary secretions are generally much increased, though there may be the opposite condition of marked dryness of the mouth. When the swelling has reached its height, pressure on the adjacent tissues causes a disagreeable sensation of tension, and chewing, swallowing, and even speaking, are at times painful and difficult. The skin over the affected part may be of a pale or of a dull-red color. Ringing in the ears and a dulling of the hearing is common. The nervous system may be affected, causing headache and delirium, or a low typhoid state may be present. The duration is about one week (six to ten days), after which time the swelling subsides, and by the tenth or twelfth day entirely disappears.

**Diagnosis.**—The diagnosis is easy, the nature and position of the swelling and the course of the disease being characteristic, while the fact that the tonsils are seldom involved prevents a diagnosis of acute tonsillitis.

Occasionally, however, in the course of septic infection or after operations, or owing to the extension of inflammation along the duct from the mouth, the parotid gland becomes the seat of an acute inflammation at first hardly distinguishable from mumps. The existence of a possible source of infection, and the fact that the gland under these circumstances usually undergoes suppuration, should lead to the recognition of the true nature of the case.

**Complications and Sequelæ.**—Mumps, as a rule, runs a mild course without any serious symptoms, but occasionally complications arise that tax the skill of the physician to the uttermost. The most common of these are *orchitis* in the male, which may be followed by atrophy of the testicle; and *mastitis*, *ovaritis*, or *vulvo-vaginitis* in the female, especially after puberty. These complications appear after the subsidence of the swelling of the glands of the neck, only occasionally developing while the glands are still affected, though cases have been reported in which the disease first manifested itself by involvement of the sexual organs. This complication lengthens the course of the attack



and increases the constitutional symptoms, but the rule is complete recovery. *Otitis media* sometimes occurs, and a lesion in the auditory nerve, with more or less deafness (which, unfortunately, may be permanent), has been observed. *Meningitis*, with active brain-symptoms, *facial paralysis*, *convulsions*, *albuminuria*, and *arthritis*, have all been noted in certain cases.

**Treatment.**—The patient should be kept in a well-ventilated room of even temperature, and in bed if the fever is at all severe, and should be isolated from those who have not had the disease. Either hot or cold applications to the swelling will often give relief, and support to the swollen gland by means of cotton and a bandage is very comforting. Saline laxatives may be given, and aconite or some simple fever-mixture at the beginning of the attack is usually indicated. These simple measures are all that are required in an ordinary case, while complications or unusual conditions must be treated as they arise.

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## TUBERCULOSIS.

**Definition.**—A chronic (less frequently acute) infectious disease, caused by the bacillus tuberculosis. This organism produces specific lesions, taking the form either of separate nodular masses or diffuse growths, infiltrating the tissues, while aggregations of these elementary tubercles give rise to large tubercular masses. Tubercles undergo caseation and sclerosis, followed in turn by ulceration (in consequence of secondary pyogenic infection), or, more rarely, calcification.

**Historic Note.**—Prior to the discovery, in the early part of the nineteenth century, by Bayle and Laennec, of the tuberculous new growth as a distinctive body, this disease had been studied chiefly from a clinical point of view. At this early period the disease was believed to consist chiefly of a suppurative process, and in its observation the physician was unaided by auscultation. Later, the tubercle was recognized as a small rounded nodule without any special histologic characteristics. Villemin in 1865 performed his epoch-making experiments, and the tubercle was no longer distinguished by its anatomic characters alone. Though the theory of the infectious nature of tuberculosis had been previously advanced by Buehl and others, it was first clearly demonstrated by Villemin's beautiful inoculation-experiments upon rabbits and guinea-pigs with particles of tubercular and cheesy substances, producing the characteristic lesions of tuberculosis. It then remained for Koch to discover (in 1881) the specific cause of the most important of all human ills—the tubercle bacillus. So soon as the specificity of the disease was definitely established it became clear that the associated inflammatory processes, that were formerly believed to be primary and to hold first place, were secondary. The important rôle, however, played by the latter, particularly in the production of the general features of the disease, will be shown hereafter.

**Geographic Distribution.**—Tuberculosis prevails in almost every

quarter of the globe, but is more prevalent in certain latitudes than in others. Thus, in general terms, it may be said to prevail less extensively in warm than in cold countries, though it lessens in frequency as we approach either pole. Local conditions, however, exercise a more decisive influence in engendering predisposition than mere geographic position. It is of quite frequent occurrence in all densely populated municipalities, and more especially in the overcrowded sections of the latter; this fact explains why the inhabitants of cities of the North are but little less spared than those of the cities of the South. On the other hand, residents of mountainous countries, owing to the purity of the atmosphere and the elevation, are rarely among its victims. The influence of *race* in predisposing to tuberculosis should also be mentioned here, the South Sea Islanders, the Indians, and the colored race being peculiarly liable.

**General Pathology of Tubercular Lesions.—Distribution of the Lesions in the Body.**—Tuberculous new growths elect, most frequently, the lung, and when the disease occurs in the adult this organ is almost invariably implicated. Next follow the larynx, intestines, peritoneum, urogenital organs, and the brain. The other chief viscera of the body (spleen, liver, heart, etc., particularly the latter) are less frequently the seat of tuberculosis. In children the lesions exhibit a different distribution, the favorite seats being the lymph-glands, intestines, bones, and joints. In them the distribution corresponds pretty closely, if we except the bronchial and mesenteric glands, to that of surgical tuberculosis.

**The Elementary (Nodular) Tubercle.**—This may be developed in any tissue to which the tubercle bacillus has found its way, and the presence of the bacillus is its sole distinguishing feature, since the self-same bodies are generated by other micro-organisms—*e. g.* certain of the worms (eggs of the distoma), actinomyces, aspergillus glaucus, and even as a result of irritation by certain foreign bodies (podophyllum). Various forms of pseudo-tuberculosis have been described, but all are due to bacteria that differ from the bacillus tuberculosis. Mallassez and Vignal described a form produced by a micrococcus occurring in a zoöglea, and this observation was later confirmed by Nocard, Eberth, and others. Charrin and Rogers have described still another form, in which they found bacilli about  $1\mu$  long, actively motile, and growing freely upon ordinary media, but not growing upon glycerin and agar, and not liquefying gelatin.

The various stages in the development of a tubercle are—

(a) *Proliferation* of the fixed-tissue elements (connective tissue, endothelium of the capillaries, etc.) of the part infected, due to the local, specific irritant action of the bacilli. These anatomic products are transformed into epithelioid and giant cells. The epithelioid cells assume various shapes, chiefly rounded and polygonal; they have vesicular nuclei, and soon show tubercle-bacilli in their interiors. A certain proportion of the epithelioid cells, as the result of increase in their size and a repeated division of their nuclei, become *giant cells*. The latter occupy the center of the tubercle, and also contain bacilli, the number of giant cells and of the bacilli being largely reciprocal. Thus, the giant cells are numerous in tubercular lymph-glands, joints, etc., in

which the bacilli are relatively few; on the other hand, they are scanty in miliary tubercles, in which the bacilli are numerous—two facts that lend support to the view held by many authors that giant cells display phagocytic action.

(b) About the site of infection a *diapedesis of leukocytes* occurs in the nature of a defensive inflammatory process. At first the leukocytes are of the polynuclear variety and are quickly destroyed; but later mononuclear leukocytes (lymphocytes) appear. These latter resist the action of the bacilli, and I think their true function is a phagocytic one. The various forms of cells described are connected and surrounded by a reticular stroma “formed by the fibrillation and rarefaction of the connective-tissue matrix” (Baumgarten).

**The fully-developed tubercles** are small, nodular bodies whose diameters range from  $\frac{1}{2}$  to 2 or 3 mm. At first they are almost transparent, but soon lose this quality in consequence of the further changes described below. They are non-vascular bodies, and invariably undergo (a) *caseation* and (b) *sclerosis*.

(a) *Caseation*.—This implies “coagulation-necrosis”—a destructive process proceeding from the center toward the periphery of the tubercle, and the result of the local action of the bacilli or their chemical secretions. The cells are thus transformed into a uniformly yellowish-gray, structureless matter. When the foci are numerous and close-set, fusion may occur, with the production of larger or smaller homogeneous masses (cheesy pneumonia). The latter may soften, resulting in the formation of cavities: this is due, usually, to secondary pyogenic infection, causing ulceration. Less frequently the cheesy masses undergo calcification or become encapsulated. Such masses may remain indefinitely and are practically harmless.

(b) *Sclerosis*.—Preceding and during the time that cell-destruction is going on in the center of the tubercles the protective forces of nature are asserting themselves, though too often without avail. In the first place, hyaline transformation, with conversion of the cellular elements into fibrous tissue, occurs. Frequently, now, the center of the tubercle is caseous and contains bacilli, while the peripheral parts are quite hard and do not contain bacilli. The fibroid change may pervade the entire tubercle. Again, the fibroid element in the tissues immediately surrounding the tubercle may be greatly increased and form new connective tissue, and this process be followed by secondary contraction, converting the tubercle into a firm fibrous nodule. The fibroid change in its completest development is observed in tuberculosis of serous membranes, especially of the peritoneum.

Whether in any given case the destructive forces, on the one hand, or the conservative, on the other, shall come off victorious depends upon several conditions. Though natural immunity is probably unknown, yet under certain circumstances and at certain times tissue-soils may successfully resist bacillary invasion. The bacilli of tuberculosis doubtless produce special toxins (*vide* Etiology), and hence there is a reasonable probability that the tissues and liquids of the body manufacture an antitoxin. The latter agent may therefore constitute one of Nature's chief means of defence. There are also soils that are moderately receptive, and these may become infected; but sooner or later the destruction



of the invading parasite may be determined by altered soil-conditions—changes induced by Nature's benign and curative efforts. It is probable that in such instances the favorable issue is sometimes to be ascribed to the fact that relatively few bacilli find lodgement, so that the average phagocytic activity and other protective processes suffice. But when the bacilli fall upon a soil that is altogether favorable to their growth their pernicious influence cannot be arrested, since the usual means that turn the scales in favor of a cure are wanting.

We are now prepared to understand the coarser appearances presented by tuberculous lesions, especially of the lungs. Fusion of minute centers of infection or of miliary tubercles results in the formation of larger nodules or areas, which lead by a process of local extension to *diffuse tuberculous infiltration* (gray infiltration of Laennec). An entire lobe may become similarly involved (tuberculous pneumonia), and "there may also be a diffuse infiltration and caseation without any special foci, a widespread tuberculous pneumonia induced by the bacilli" (Osler).

The term "gray infiltration" is misleading from a pathologic point of view, since the morbid changes differ in no essential manner from those described as occurring in the miliary or nodular tubercle. Moreover, the latter also presents a grayish appearance. The apparent difference between a miliary tubercle and diffuse tubercular infiltration lies in the fact that the latter displays a greater tendency to spread by direct extension.

**Associated Inflammatory Processes.**—The tubercle bacilli excite associated inflammatory processes in the organs affected, and if the tuberculous lesions run a slow course, a limited wall of true fibroid induration circumscribes the area involved. By means of this induration the natural protective forces, either temporarily or permanently, check the progress of the local lesions, and the change is strictly analogous to the sclerosis that takes place in the peripheral parts of the elementary tubercle or immediately surrounding the latter, as in tuberculosis of serous membranes. On the other hand, when the tuberculous infiltration is less tardily developed the secondary inflammatory processes may show changes similar to those of catarrhal or croupous pneumonia. It is a noteworthy fact that the constitutional features in tuberculosis are not so much dependent upon the primary as upon a secondary infection, chiefly with the streptococci. The latter are responsible for the serious septic element in the various varieties of tuberculosis (especially pulmonary), and some contend that the tubercle bacilli can excite suppuration directly. The pus, however, in this instance does not contain the streptococci, and is sterile. Mixed infection is, I believe, the rule (*vide* Pathology of Pulmonary Tuberculosis).

**Etiology.**—The Specific Cause and its Physical Characteristics.—In 1881, Koch discovered the tubercle bacillus, which is the sole bearer of the disease. This bacillus is rod-shaped, straight or somewhat bent, and slender, its length equalling about one-third or one-half of the diameter of a red blood-corpuscle (Fig. 24). Its extremities are slightly rounded, it is non-motile, and on the interior of the bacilli small colorless spots can be observed on microscopic examination; these clear spaces in the bacilli represent plasmolysis, and have nothing to do with spore-formation. Spores undoubtedly do occur, but have not yet been demonstrated.

When stained the bacilli have a somewhat beaded appearance, this being probably due to slight bulgings caused by the presence of spores. The tubercle bacillus is one of the few varieties of bacteria that retain the anilin dye after washings with acids.

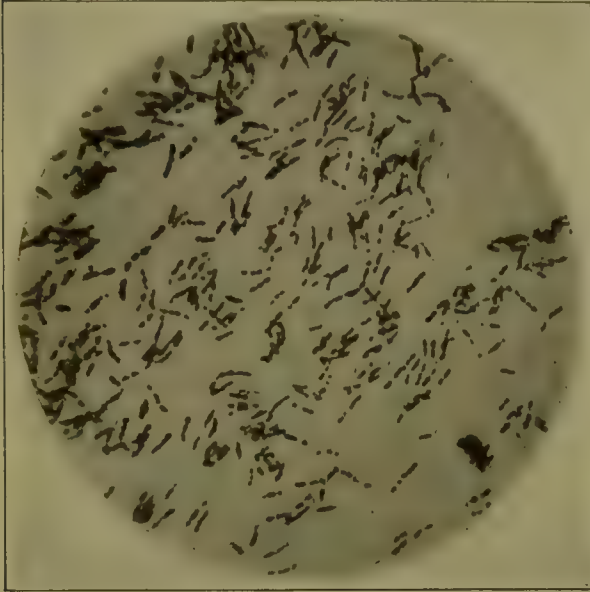


FIG. 24.—Tubercle bacillus in sputum (Fränkel and Pfeiffer).

**Biology.**—The bacilli can be grown on culture-media, but not without difficulty, since they demand an even temperature between 98° and 100° F. (37.7° C.), or that of the human body. The best soil is blood-serum previously coagulated by heating. Over the latter may be gently rubbed tuberculous tissue, which is then allowed to remain on the surface. The growth of the bacilli requires about two weeks, when colonies appear as dry, grayish-white or gray-

ish-brown, thin scales or masses on the surface of the culture-medium. From such cultures others may be grown on glycerin-agar or on the potato.

**Inoculations** into the guinea-pig and other animals are succeeded in two or three weeks by the appearance of elementary tubercles—first, locally, and then in other organs of the body.

**Chemical Products.**—The growth of the bacilli is probably attended by the formation of secretory products. Thus an albuminoid substance has been separated, and this when injected into the body of an animal produces fever, lasting a day or two. The albuminoid separated for cultures of tubercle bacilli is a nuclear proteid, and not a specific toxin. There have also been isolated a ptomain and, some contend, an extract which displays pyogenic properties (Koch's tuberculin). The constitutional features of the disease may be ascribed, in part, to the circulation of these poisons in the blood, but they are probably infinitely less important in this direction than the products of suppuration.

**Sources of the Bacilli.**—The chief source of the bacilli is the sputum of tuberculous patients. It has been shown that in the advanced stage of pulmonary tuberculosis several billions of bacilli are expectorated daily (Nuttall), and the desiccated sputum is wafted into the atmosphere in the form of dust-like particles containing innumerable bacilli. When the facts that tuberculosis is almost universally prevalent, and that each patient throws off countless millions of bacilli are remembered, it is clear that abundant opportunity is everywhere presented for infection, or, in other words, that secondary sources of infection are numerous and varied.

**Distribution of the Bacilli.**—The tubercle bacillus is exceedingly tenacious of life, this being its chief distinguishing characteristic. Hence it is found in a viable condition, both (a) inside and (b) outside of the body.



(a) *Inside of the Body*.—As before stated, the number of bacilli found in tuberculous growths varies within wide extremes. In general terms, it may be said that the more rapidly the process advances the greater the number of parasites present. It must not be forgotten, however, that the activity of the tuberculous processes is intimately connected with the degree of resistance offered by the tissues. A chronic tuberculous focus may establish a fistulous connection with a vein or a lymph-vessel, and thus scatter the bacilli to the remotest parts of the body; and in such instances (as the direct effect of the original number of bacilli present) a chronic is quickly converted into an acute form of tuberculosis. The bacilli may also be found in the bodies of non-tuberculous persons. Strauss<sup>1</sup> demonstrated virulent bacilli within the nasal cavities of healthy persons whose positions necessitated their association with, and frequent presence in rooms occupied by, tuberculous patients.

(b) *The Bacilli Outside of the Body*.—Tubercle bacilli can maintain their existence almost indefinitely outside the body. On the other hand, they probably do not develop or multiply under the usual external influences, though, as I have said, their vitality is extraordinary. Their destruction cannot be effected by freezing nor by desiccation, and they survive for months in water. Their power to resist chemical agents (nitric acid, etc.) is also very great, but they may be destroyed by boiling for four or five minutes or by exposure to the direct solar rays. Tubercle bacilli are undoubtedly present in all inhabited places, and it is obvious that they may be conveyed for long distances by means of water, milk, and in many other ways.

The *sputum* dries and flies into the atmosphere in the form of dust, which not only floats in this medium, but also settles upon articles of furniture, the floor, the walls of living-rooms, hospital wards, draperies, clothing, bed-linen, etc.; and from these resting-places it may be conveyed back into the atmosphere. It has been shown, experimentally, that the dust obtained from the walls or from the air of rooms and hospital wards occupied by tuberculous patients is frequently, though not invariably, infected. It is the *in-door atmosphere*, laden with bacilli, that is especially liable to excite tuberculosis when breathed more or less constantly. In places only rarely frequented by consumptives the dust is usually free from virulent bacilli.

**Modes of Infection.**—(1) *Inhalation of the Bacilli*.—In the vast majority of instances the bacilli are inhaled with the inspired air, but it is important to recollect that the exhaled breath of tuberculous patients is not infectious. It is the dried sputa floating in the atmosphere that are pathogenic; and occasionally, when infection occurs in this manner, the bacilli may attack first the upper respiratory passages, producing primary tuberculosis of the larynx and nose. Almost invariably, however, primary infection takes place in the *smaller bronchi*, or less frequently in the *lungs*; and that these are the points of election is shown by the fact that healed tuberculous lesions are often met with on post-mortem examination in the bronchi and lungs of persons who died of other diseases. Under similar circumstances the *bronchial glands* may be found to present tuberculous lesions. Thus, in 8 out of 30 cases in

<sup>1</sup> *Münchener medicinische Wochenschrift*, Munich.



which both old and recent tuberculous lesions were absent H. P. Loomis found the bronchial glands infective to rabbits.

It is obvious that the bacilli which cause fresh cases come indirectly from other tuberculous subjects, and it has long been supposed that tuberculosis is a contagious affection; unlike small-pox, scarlatina, and other acute contagious diseases, however, tuberculosis is not transmitted by a single contact with a person ill of the disease. On the other hand, Flick and others have shown that persons who come into contact with, or who live in close proximity to, affected persons frequently fall victims to the same affection. ("The latter is as truly contagious as the former, differing only in degree.") We can safely say, therefore, that, though less liable to be transferred by contact than certain other affections, yet on account of the fact that tuberculosis usually pursues a chronic course there is every opportunity for prolonged or repeated contact with resulting infection.

Flick's elaborate topographic study of phthisis in the Fifth Ward of the city of Philadelphia, extending over a period of twenty-five years, shows conclusively that consumption obeys the laws of infectious and contagious diseases. His researches furnish incontestable proof that the tuberculous virus is limited to centers, and that the latter owe their existence to previous cases in the same house or locality; that a house which has had a case of consumption will probably have others within a few years, and may have a very large number of cases in rapid succession; and that approximate houses are considerably exposed to the contagion.

The *contagious theory* of tuberculosis gains support from the fact that husbands have been frequently observed to contract the disease from their wives, and the latter, since they are more constantly confined in the house, to become infected yet more frequently from the former. Weber has observed the case of a tuberculous husband who lost four wives in succession, another who lost thrée, and four others who lost two each. In like manner, the statistical studies of Cornet, Niven, Baer, and others show that the disease spreads through factories, prisons, cloisters, and even among the physicians, nurses, and attendants in hospitals for the reception of tuberculous patients, producing a mortality-rate from this disease ranging from 45 to 75 per cent. Seventy-three per cent. of nurses up to the age of fifty die of tuberculosis (Whittaker). It is obvious that those who are engaged in making the beds, dusting and sweeping the rooms of patients, are most exposed; and on the other hand, better hygienic living among these classes of individuals, and improved hygienic arrangements in prisons, institutions, and hospitals, have been found to reduce, decidedly, the death-rate from this dread affection. This result is to be accounted for by the following facts: (a) There is thus established a greater tissue-resistance to the bacillus tuberculosis on the part of the persons exposed; and (b) The germs are thus to a greater extent disseminated. Obviously, then, in institutions in which the proper sanitary precautions are used there may be few if any instances of communication by contagion; and from the records of the latter, facts opposed to the contagious theory of the disease can readily be furnished.

(2) **Infection by Swallowing.**—(a) That the milk of tuberculous ani-

mals contains the bacillus, and that the use of contaminated milk may infect the human subject, are well-established facts.<sup>1</sup> Gerlach and Klebs long since observed the occurrence of the disease in animals fed with milk from cows affected with the so-called "pearl disease." It is not even necessary that the animal infected should have tuberculous mammitis (Ernst), though some are of contrary opinion (Flick, Sidney Martin, and others). The exact frequency of this mode of infection is not known, but there is some little clinical evidence to support it. Infected animals, especially cows and pigs, that suckle their young very frequently transmit the disease to the latter, the infection usually resulting in intestinal and mesenteric tuberculosis. Hence it is obvious that the bacillus of tuberculosis is, in this instance, swallowed and finds lodgement in the *primæ viæ*. Bang has even shown that butter made from the milk of tuberculous cows may be infectious. It is entirely analogous in the human race, bovine and human tuberculosis being one and the same affection; and hence the tuberculous mother is likely to transmit the disease to her suckling offspring. This explains, adequately, why abdominal tuberculosis is frequent in children.

(b) *The meat of a tuberculous animal* may rarely be infectious, but the bulk of experimental evidence would seem to show that, unless the parts consumed are the seat of tuberculous deposit, infection does not follow. The authentic instances that have been recorded in which human tuberculosis was the result of the use of infected meat are rare indeed; but that this is a possible source of tuberculous infection in man must not be forgotten. Again, the possibility of contamination during the course of preparation for the market, as well as during its transportation, must also be recollected. The experiments of Aufrecht, Chauveau, Klebs, Trappeiner, Parrot, and others show that tuberculosis may be communicated by incorporating with the food the expectoration from tuberculous patients.

(3) **Infection by Inoculation.**—Tuberculosis may be transferred by direct inoculation, as shown originally by Villemin's beautiful experiments upon the eyes of guinea-pigs. Infection may take place, though this is rare, through slight cutaneous lesions (cuts, fissures, excoriations, etc.), but only as the result of accidental inoculation of tuberculous matter. In this manner there is produced a local tuberculosis of the skin, as a rule. Rarely, the contagion is conveyed by the lymphatics to the glands in the vicinity of the point of infection. Persons who follow certain occupations are more or less liable to this mode of infection—*e. g.* butchers, handlers of hides, dissectors of dead bodies, and, rarely, surgeons.

Tuberculous virus may be introduced into the tissues through any open lesions and the characteristic local change follow; hence quite rare instances of transmission by inoculation occur in divers ways (the bite of a consumptive, a cut from the broken spit-glass of the latter, or even from his pocket-knife, as I have seen in one instance).

The handkerchiefs, body- and bed-linen of the patient may infect by inoculation those who handle or wash them frequently, if they chance to have a fissure or excoriation upon the hand. No doubt *lupus* also

<sup>1</sup> See the elaborate statistical studies of Dr. George Cornet: "Die Tuberkulose in den Strafanstalten," *Zeitschrift für Hygiene*, Bd. x., 1891.



arises in the same way. Czerny has reported 2 cases of infection by transplantation of the skin; Collings and Murray, 3 cases by tattooing (?). The contact of the lips of tuberculous operators with surgical wounds (as in sucking the latter) may be the means of transmitting the disease; in this way tuberculosis may be, and undoubtedly has been, communicated during the performance of the rite of circumcision.

(4) **Direct Hereditary Transmission.**—In exceptional cases the bacillus is found in the fetus *in utero*. In such instances the disease may remain latent, to break forth during childhood or later in life; and even though the fetus itself may display no evidence of tuberculosis, the fetal viscera may yet be infective to guinea-pigs (Birch-Hirschfeld). Lehmann<sup>1</sup> has reported an undoubted instance of intra-uterine infection. The tuberculous mother died of tuberculous meningitis three days after the birth of her child, and the child lived twenty-four hours. In its spleen, lungs, and liver were found nodules resembling tubercles and containing tubercle bacilli in large numbers. Galtier has inoculated a pregnant animal with the disease, and found that the offspring was, in consequence, tuberculous at birth. The views of Baumgarten upon this question should be accorded careful consideration. This author believes that the contagion may be transmitted and become pathogenic at a variable period after birth—first, because the affection is very frequent in young children, even during the first months or weeks of life; and, secondly, because certain structures, not apt to be accidentally infected, are commonly the seat of tuberculous lesions in children—the bones and joints. He states that hereditary infection may occur in three ways: by the passage of bacilli through the placenta; by infection of the ovum from the internal tissues or fluids; and by infection carried in the fructifying sperm. After birth the bacillus may at any time either lose its vitality or take on a luxuriant growth. It is not known, however, in what percentage of these cases the lungs, intestines, peritoneum, and lymph-glands are free from tuberculous lesions.

Again, there are certain interfering conditions that must be borne in mind. Of these, two deserve to be emphasized: (a) the fact that a child born of tuberculous parents is more receptive to the tubercle bacillus than one born of healthy stock, and (b) that it is more liable to accidental infection, as by swallowing the virus (particularly if breast-fed) or by inhalation.

The instances of direct transmission that have been traced definitely have occurred through tuberculous mothers. The observations of Csokor<sup>2</sup> upon hereditary tuberculosis in cattle also corroborate this dictum; but as the result of carefully conducted experiments by Vignal<sup>3</sup> it is reasonably certain that invasion by heredity is very rare.

**Predisposing Causes.**—(1) **Race and Nationality.**—The effect of nationality upon the receptivity to tuberculosis can be studied advantageously in America on account of the cosmopolitan character of the population. The tuberculous tendency on the part of Indians of this continent, among whom the death-rate from this disease is not less than 25 per

<sup>1</sup> *Berlin. klin. Woch.*, July 9, 1895.

<sup>2</sup> *Deutsche medizin. Zeitung*, Berlin, Jan. 29, 1892.

<sup>3</sup> *La Semaine médicale*, Paris, Aug. 1, 1892.



cent., even in the most favorable climates, has been observed repeatedly, and the fact that the negro race is highly receptive to tuberculosis is also well known. Osler<sup>1</sup> gives the following corroborative statistics: "Of the 427 cases of pulmonary tuberculosis at the Johns Hopkins Hospital for the two years ending June 1, 1891, there were 41 cases in the colored—*i. e.* about 1:10. The ratio of colored to white of all patients in the wards has been 1:7." Sears<sup>2</sup> found that in 200 cases of tuberculosis nearly 50 per cent. belonged to the first and second generation of Irish immigrants. Such facts serve to show that differences unquestionably exist as regards certain nationalities.

(2) **Hereditary Predisposition.**—The percentage of cases in which heredity can be traced has been variously estimated at from 10 to 40. As before intimated (*vide Direct Hereditary Transmission*), a child reared by tuberculous parents runs great danger of being infected accidentally; and again, a person living in an infected house (with or without the presence of a tuberculous patient) is very liable to become infected, whether his antecedents give a tuberculous history or not. It follows that a correct estimate of the number of cases of phthisis in which hereditary influence plays an etiologic part cannot be obtained. Too much importance has heretofore been attached to the influence of inherited constitutional peculiarities to the exclusion of other potent factors. Moreover, the latter may be acquired as the result of certain debilitating influences (childbirth, defective food-supply, close living- or working-rooms, etc.). An inherited tendency to tuberculosis is more unfailingly transmitted through the mother than the father. Children begotten of parents who are drunkards, or who suffer from certain chronic incurable diseases (syphilis, cancer, etc.) at the time of the birth of their children, are liable to inherit a condition of the system which renders them peculiarly liable to tuberculosis, unless the tendency is overcome by a proper environment, together with systematic physical training, during the first years of life. Moreover, persons who have the so-called tuberculous diathesis are frequent sufferers from catarrhal affections, especially of the respiratory organs. The latter condition forms a marked predisposing factor; yet, on the other hand, tuberculosis is met with in persons who are robust and have apparently well-formed chests and lungs.

The older authors of medical text-books describe two types of conformation—the *tuberculous* and the *scrofulous*. The latter has a heavy figure, thick lips and hands, large thick bones, and an opaque skin; the former, a light figure, bright eyes, thin skin, oval face, and long, thin bones. The phthisical type of the chest will be referred to in connection with the physical signs of pulmonary tuberculosis. In this connection emphasis should be given to Cohnheim's view, which is for the greater part correct, to the effect "that the so-called phthisical habit is not an indication of a tendency to, but actually of the existence of, tuberculosis." Whilst the recognition of the tubercular diathesis has its practical bearing, it must be recollected that the term implies merely a "delicacy of constitution, incomplete growth, and imperfect development" (Fagge).

<sup>1</sup> *Text-book of Medicine*, p. 204.

<sup>2</sup> *Boston Medical and Surgical Journal*, April 4, 1895.

(3) **Previous Infectious Diseases.**—That there is no tendency to the transition of other diseases into tuberculosis, as was formerly supposed, cannot now be questioned in view of the undoubted specific nature of the latter disease. Tuberculosis is, however, embraced among the sequelæ of such affections as acute infectious and chronic diseases—influenza, measles, whooping-cough, typhoid fever, cirrhosis of the lungs, and diabetes mellitus (the latter disease involving a predisposition to the former)—for the reason that they render the tissue-soil, especially that of the respiratory tract, more favorable to tuberculous infection. It seems proper to mention here the fact that certain other diseases display an antagonistic effect (chronic valvular disease, pulmonary emphysema, etc.). Our knowledge of the subject is as yet quite incomplete, but of an affection which occurs so very frequently and carries off so large a percentage of the population as tuberculosis a more accurate and extended knowledge would be welcomed by the profession.

(4) **Age.**—This affects predisposition decidedly, though tuberculosis may occur at any or all times; and the relation between age and the distribution of the lesions has been previously indicated. Certain forms of tuberculosis are especially frequent in young children (meningeal, mesenteric, and lymphatic).

Pulmonary tuberculosis is most common between twenty and thirty. It is more rare during early childhood and in the aged, but may appear at any period of life, and the cases that occur in young children are apt to be more rapid in their progress.

(5) **Sex.**—Predisposition has but slight relation to sex. Females are, however, somewhat more liable than males, and pregnancy in particular is a disposing factor. Again, when tuberculous females become pregnant the progress of the affection is accelerated, and even more so by the period of lactation. Regarding tuberculosis as being pre-eminently a house-disease, females are more exposed to contagion than males, because they are more closely confined in-doors.

(6) **Climate and Soil.**—Humidity of the soil and abundant atmospheric moisture increase the prevalence of tuberculosis. It is especially common in regions where sudden variations of temperature, or protracted cold with dampness, prevail. This increase is most probably associated with a heightened vulnerability, due to an increased tendency to catarrhal affections of all kinds (Osler). It has been shown that proper drainage of marshy districts has diminished, to some extent, the frequency of this disease (Buchanan), and, on the other hand, mountainous districts are often remarkable for freedom from the disease.

**Local Causes.**—(1) **Occupation.**—Persons whose employment exposes them to different forms of irritating inhalations are particularly liable. In such, however, there is usually first developed a fibroid induration, and the latter in turn is followed by pulmonary tuberculosis. The continual inhalation of an atmosphere laden with noxious particles, such as is met with in ill-ventilated and overcrowded working or living apartments, renders the tissue-soil more vulnerable.

(2) **Bronchial Catarrh.**—An acute catarrh of the small bronchi prepares the soil for tuberculous infection. Frequently, however, this is the first step in tuberculosis, since the latter disease almost invariably begins as a local catarrhal process, involving the smaller apical bronchi.



The fact may here be pointed out that gastro-intestinal catarrh increases the bodily receptivity for tuberculosis.

(3) **Tubercular Pneumonia.**—In like manner, pulmonary tuberculosis may follow an unresolved pneumonia, but such cases are, as a rule, instances of tuberculous pneumonia primarily.

(4) **Hemoptysis.**—According to some authors, hemoptysis is potent in producing pulmonary tuberculosis. It is, however, certain that in most instances in which it appears to precede phthisis, and have a causal connection with it, it is in reality a symptom of existing pulmonary tuberculosis.

(5) **Pleurisy** may be, though rarely, the starting-point of phthisis. Its predisposing effect may be attributable to compression of the lung, thus interfering with the respiratory excursions, or to the bronchitis which is frequently associated. Pleurisy sometimes initiates fibroid induration, which may then terminate in a tuberculous affection; but the fact is to be emphasized that a very large proportion of the cases of apparently primary pleurisy are tuberculous in nature.

(6) **Intrathoracic Tumor.**—Tuberculosis is often associated with intrathoracic tumors, and especially with aneurysm. Fehde<sup>1</sup> has reported 3 interesting cases of the kind.

(7) **Congenital or acquired contraction of the orifice of the pulmonary artery** predisposes markedly to tuberculosis.

(8) **Trauma.**—Injuries to the chest-wall, with or without ulceration of the lung, are frequently followed by pulmonary tuberculosis. The explanation of this association is to be found in the fact that trauma increases largely the susceptibility of the parts injured by diminishing phagocytic activity—the natural power of resistance. It is a familiar observation in surgical practice that after injuries to, or operations on, joints, tuberculosis commonly ensues. Again, operations upon tuberculous lesions are succeeded by general tuberculosis—often acute—in about 8 per cent. of the cases.

## TUBERCULOSIS OF THE LYMPH-GLANDS.

(*Scrofula.*)

Scrofula implies tuberculous infection, and scrofulous material inoculated upon susceptible lower animals, especially guinea-pigs and rabbits, invariably causes tuberculosis. The virus is, however, less virulent than that derived from other sources, and this explains the slow progress and often latent character of tuberculosis of the glandular system. A major predisposing factor is *age*, this form of tuberculosis preponderating in children. Hecker, from an examination of the records of the Munich Pathological Institute, found that in 147 cases of tuberculosis among children the lymphatics were affected in 92 per cent.; and it is generally conceded that in young adults tuberculous adenitis is not uncommon, and that it is rarely met with during and after the middle period of life. The lesions generally remain limited to the glands first infected—*i. e.* the cervical, mesenteric, etc., as the case may be—and this for the reason that the natural powers of resistance in the tissues are often able

<sup>1</sup> "Lungentuberculose mit Brusthöhlengeschwulste," *Inaug. Diss.*, Leipzig, 1894.



to oppose the march of the destructive forces. Another predisposing condition is an acute or chronic catarrh of the mucous membranes.

The cases are all divisible into two groups: (1) Local tuberculous adenitis, and (2) general tuberculous adenitis.

(1) **Local Tuberculous Adenitis.**—(a) **Cervical.**—This is the most frequent form, and is especially common among children. Of 2035 persons examined by Valland, enlarged cervical glands were found between the ages of seven and nine in 96 per cent.; between ten and twelve in 96.1 per cent.; between thirteen and fifteen in 84 per cent.; between sixteen and eighteen in 69.7 per cent.; and between nineteen and twenty-four in 68.3 per cent. Tubercle bacilli were found in the cervical lymph-glands in about 68 per cent. of adults. Negroes are found to be more prone to the affection than whites.

*Etiology.*—I have stated before that tubercle bacilli are sometimes found on the nasal mucous membrane of healthy persons. The presence of an acute or chronic catarrh of the nasopharynx may now lower the resistance of the tissue-cells, so that the bacilli may gain access to the lymph-current, and through the latter to the neighboring glands, setting up tubercular adenitis. Though often the seat of tubercular invasion, the cervical lymph-glands do not furnish a highly favorable soil for the growth and development of the bacilli, and hence the tendency toward latency of tuberculous disease of these organs.

The *tonsils*, owing to their free communication with the atmosphere, in which there is a wide diffusion of tubercle bacilli, may be primarily infected. But here also, as in the case of other glandular structures, there is a tendency for the affection to become latent, for the reason that the tissue-soil after a prolonged contest generally gains the ascendancy over the invading bacilli. The latter may, however, under certain favorable conditions, break down the barriers opposed by nature and effect a lodgement elsewhere, or even become widely diffused through the economy. Thus Kinckmann in 64 autopsies found 25 cases of tuberculosis, in 12 of which the tonsils were affected.

A third mode of infection of the cervical lymph-glands is through the medium of slight injuries and abrasions of the skin or certain forms of skin-eruptions (eczema, etc.). These serve as doors of entrance for the bacilli, which find their way into the neighboring lymph-glands through the lymph-channels. Compared with infection from within, this mode is most probably much less frequent.

*Symptoms.*—The main feature is a visible enlargement of the affected cervical glands, chiefly the submaxillary. At first the glands are too small to be even palpated; later, they can be felt as small, firm tumors underneath the skin. By and by they appear as visible protuberances, ranging in size from that of an English walnut to that of a hen's egg or even larger. The skin over the enlarged gland is freely movable, as a rule; less frequently it becomes adherent—an indication of suppuration. When an abscess forms and is allowed to open spontaneously, there remains a chronic discharging sinus. Suppuration is attended with fever, anemia, and emaciation. In well-marked cases the separate tumors coalesce, forming large and irregular masses. The affection is usually bilateral, though almost invariably it is more marked on one side than on the other.

Not infrequently, in addition to the enlargement of the submaxillary, post-cervical, and supraclavicular glands, there is also involvement of the axillary, as was the case in a fatal instance in my own practice. The patient was a male child, eight years of age, who developed pulmonary tuberculosis. In such instances it may reasonably be assumed that the bronchial glands also become implicated, and frequently become the exciting cause of the lung-tuberculosis.

The *diagnosis* is based upon the history, the associated evidence of the tuberculous diathesis (keratitis, conjunctivitis, eczema of the scalp or face, nasopharyngeal or bronchial catarrh, etc.), together with the enlargement of the superficial cervical glands. Bacilli have occasionally been found in the purulent discharge from abscesses.

The *course* of this affection is exceedingly slow, often extending over a number of years. Many cases, however, recover if surgical interference be employed. On the other hand, neglected cases are a menace to the life of a patient, since they may be followed by diffusion of the bacilli, with the development of a fatal form of disease.

(b) **Bronchial.**—Tuberculosis of the bronchial glands may be primary, or secondary to infection of the lungs, and it is commonly preceded by or associated with bronchial catarrh, which is its chief predisposing cause. The primary form is met with frequently in young children, the mediastinal lymph-glands being affected uniformly in 127 cases at the New York Foundling Hospital (Northrup).

The bronchial and tracheal glands are the receptacles for all foreign substances, including the tubercle bacilli that are not dealt with by the broncho-pulmonary phagocytes. After infection with tubercle bacilli the lymph-glands become swollen, tumefied, and are the seat of caseous change; later they may undergo calcification or proceed to abscess-formation. The latter may rupture either into the lungs, into the trachea or the bronchi, or into a pulmonary blood-vessel.

*Symptoms.*—If a fistulous communication be established with the air-passages, cough and expectoration of purulent material, blood, and caseous matter containing bacilli will be noted.

*Secondary infection* of the lung may occur in this manner. When rupture takes place into a vessel systemic infection promptly follows. Tubercular adenitis involving mediastinal lymph-glands may also lead to infection of the pericardium and then proceed to tuberculous pericarditis.

(c) **Mesenteric** (*Tabes Mesenterica*).—This may be primary or secondary, the latter being very common and a secondary infection to intestinal tuberculosis.

The former is rare, however, and the intestinal catarrh with which it is associated is doubtless tuberculous in the vast majority of cases. The mode of infection has already been pointed out. The lesions presented are similar to those met with in tuberculous bronchial glands.

The *symptoms* are not always distinctive, and may be entirely negative during the life of the patient; hence the condition is often incidentally discovered during the post-mortem examination. The local symptoms when marked are due in the main to an associated peritonitis. The abdomen is painful and more or less swollen. Peritoneal effusion is present, and sometimes sufficient in amount to be detected by the cus-



tomary physical signs. Large and small nodules may sometimes be felt. Diarrhea is a marked and obstinate feature and is usually due to tuberculous intestinal ulcers. Fever of an intermittent type is almost constantly present, causing emaciation, and the objective changes (pallor of skin, mucous membrane, etc.) due to anemia become pronounced. This form of tuberculosis may persist as a local condition, but there is danger of extension to other organs (pleura, lungs, etc.). On the other hand, in the adult pulmonary tuberculosis may be followed by involvement of the mesenteric glands without involvement of the intestines, and in such instances there occurs an extension by contiguity along the course of the lymphatics that pass through the diaphragm, and finally, in adults, primary tuberculous new growths may be met with in the mesenteric glands.

*Diagnosis.*—A probable diagnosis can usually be made if careful attention be paid conjointly to the symptoms, physical signs, and course of the affection. The detection in a child of a tumor which may be moderately hard, doughy, or even fluctuating will aid materially in the diagnosis, and will also afford evidence of tuberculous disease in other organs.

(2) **General Tuberculous Adenitis.**—This term implies tuberculous disease of the lymph-glands throughout the body, with little if any involvement of other organs; this is a rare condition. The affection may begin as a local tuberculous lymphadenitis, nearly all of the rest of the glands of the body becoming secondarily implicated. The primary seat of the trouble is perhaps most frequently the cervical lymph-glands, though in one instance observed by myself the mesenteric glands first became affected, the case terminating in pleuro-pulmonary tuberculosis.

*Symptoms and Diagnosis.*—There is protracted fever, the temperature being of the remittent or intermittent type. Wasting and debility are progressive until the patient presents a decidedly puny aspect, while the lymph-glands that are accessible to inspection and palpation are more or less enlarged and manifest a marked tendency to suppuration. The affection is usually chronic, though very exceptionally it may exhibit an acute course. One of the chief dangers overhanging the sufferer in this affection is that, owing to liberation of the bacilli, the meninges or the lungs may become tuberculous; these cases may also terminate unfavorably from asthenia. Cases in which the glands are but little enlarged, while the general features are marked, are puzzling. On the other hand, when the superficial lymph-glands are greatly enlarged the affection may bear a striking resemblance to Hodgkin's disease.

### ACUTE TUBERCULOSIS.

This form of tuberculosis is characterized anatomically by the rapid development of miliary tubercles in many and widely-separated parts of the body. In some instances the new growths are pretty evenly distributed through all the organs of the body, manifesting the clinical symptoms of an *acute general infection*. In other instances there is a tendency to centralization of tuberculous growths, as, for example, in the lungs (pulmonary variety) or in the meninges of the brain and spinal cord (meningeal variety).



**Pathology.**—The fact is to be emphasized that somewhere in the body there is an old tuberculous focus. Apart from this primary lesion, the anatomic changes consist in the widely disseminated miliary tubercles. Their most frequent seats are the lungs, liver, and spleen; less frequently, the marrow of the bones, the heart, the choroid, and the meninges. In some of the organs, particularly the meninges, lungs, etc., the tubercles may be readily perceived by the naked eye, while in others they frequently cannot be detected without the aid of the microscope. It must not be forgotten that in some of the more protracted cases the nodular tubercles may grow into foci of considerable size, ranging from that of a lentil to that of a pea.

**Etiology.**—This has been, in the main, given in connection with the general etiology of tuberculosis (*vide supra*), though a few special points remain to be adduced. The acute forms of tuberculosis are decidedly more frequent during infancy and childhood than during adult life, and with few exceptions the cases are secondary to a local tuberculous focus in one or more lymph-glands (tracheal, bronchial, mesenteric, etc.) or in the lungs. More rarely a pre-existing tuberculous focus in the kidneys, the bones, or the skin may give rise to the affection, as may the occurrence of certain other acute infectious diseases—such as measles, whooping-cough, and influenza, in children, and typhoid fever and lobar pneumonia, especially with delayed resolution, in adults.

**Modes of Infection.**—Most frequently there is established a fistulous connection between the local tuberculous focus and a vein. The tubercle bacilli thus find their way into the circulation, and general infection promptly follows. This occurs in a great proportion of the cases in the thorax, a pulmonary vein being opened in such instances. A second mode of infection, though decidedly more rare than the above, is the rupture of a tuberculous focus into the thoracic duct, in which case the tuberculous material passes almost directly into the subclavian vein. In these cases, according to Ponfick, the disease is less rapid in onset and less acutely evolved.

**Clinical History.**—That miliary tubercles may exist in many organs of the body (liver, heart, etc.) without giving rise to symptoms is a noteworthy fact. Cohnheim and Manz have discovered miliary tuberculosis of the choroid when the condition was only detectable with the aid of the ophthalmoscope.

The following forms of the disease may be distinguished:

#### GENERAL MILIARY TUBERCULOSIS.

##### TYPHOID FORM.

The **symptoms** are those of a general infection of the body, there being in most cases a period of incubation, during which the patient complains of malaise, headache, chilliness, feverishness, and increasing debility. Rarely, the onset is comparatively sudden. The reaction of the nervous system against the poison, which is now scattered to all parts of the body, is shown by such symptoms as the fever, which rapidly increases, a rapid, feeble pulse, and mental dulness or delirium. The tongue becomes dry, and sometimes also brown. The respirations are accelerated, and there is more or less cyanosis, with which symptom is

associated a peculiar and characteristic pallor of countenance. Coincidentally with the febrile exacerbations the cheeks may wear a circumscribed blush. Among the rarer early symptoms is epistaxis. The patient soon becomes profoundly prostrated or experiences a feeling of anxiety: if, as sometimes happens, the course is protracted, weakness, anemia, and especially emaciation, are well marked and assume diagnostic importance. These cases sometimes pass into the pulmonary or the meningeal form, the patients often succumbing speedily to such localized developments.

*Fever.*—The temperature usually pursues a high range, although there are not a few cases in which the entire course is afebrile. Again, it occurs not infrequently that the temperature is normal or nearly so for a short period. The usual temperature-curve ranges at first between 102° and 104° F. (38.8°–40° C.), and then continues to rise, with the development of the serious general condition in a way exactly similar to that observed in typhoid fever. In many instances the fever is irregularly remittent, at least at intervals, if not so constantly. Thus, periods of irregular fever may alternate with others of continued, and later deeply remittent or distinctly intermittent, fever.

*Nervous Symptoms.*—In most cases the nervous symptoms are not prominent. In a smaller number headache, vertigo, delirium, and often stupor, become marked at an early stage and may persist. They are due to the general infection.

*Circulatory System.*—The pulse is small, and its rate is out of proportion to the fever, varying from 100 to 140 or higher. It may become irregular, particularly if the meninges be involved.

*Respiratory System.*—The breath is somewhat hurried and labored; there is a cough, but it is not annoying as a rule; and there is a slight expectoration, which is not characteristic. If there be present simultaneously in the lungs an old tuberculous focus, the expectoration may be more profuse and typical. The bacilli are also absent from the sputum unless an old tuberculous lesion pre-exist in the lungs.

**The physical signs** are those of a moderate, diffuse bronchitis, though local signs of consolidation or pleurisy may develop late in the course of the affection. On the other hand, such signs may be evidences of an old tuberculous affection.

*Digestive System.*—As before noted, there are anorexia and a dry tongue (symptoms due to the systemic infection), while vomiting may occur at the outset and excessive thirst is common. The spleen usually becomes enlarged, though only to a moderate extent.

*Ocular Symptoms.*—The important symptom presented by the eye is the presence of choroid tubercles, which may be determined by a careful ophthalmoscopic examination. Their absence does not militate against the diagnosis of general miliary tuberculosis, since they may be too few to be detected, or possibly absent altogether. Their demonstration is always exceedingly difficult, and only possible with the skilled ophthalmologist.

**Diagnosis.**—This form of tuberculosis is often with difficulty discriminated from typhoid fever, but in the following table I have endeavored to contrast points of dissimilarity:

## ACUTE GENERAL MILIARY TUBERCULOSIS.

Family history of tuberculosis, or presence of a pre-existing focus.  
Less characteristic.

Absent.

Curve of a decidedly irregular type.

Pulse rapid, out of proportion to fever.

Much increased and labored.

Face dusky, with peculiar pallor.

Abdominal symptoms are not suggestive.

No characteristic eruption.

Present or absent according to involvement of meninges.

Leukocytosis present (if there be suppuration).

Choroid tubercles may often be detected.

Tubercle bacilli rarely demonstrable in the blood.

Very exceptional.

Absent.<sup>1</sup>

## TYPHOID FEVER.

Coexistent with an epidemic or following previous cases of typhoid.

Evolution of the disease is characteristic.

Epistaxis a common early symptom.

Temperature-curve of the continued type.

Pulse often dicrotic; slow in proportion to fever.

Respiration moderately increased.

No duskiness of face.

Abdominal symptoms (stools, enlarged spleen, tympanites, etc.) suggestive.

The eruption (appearing in successive crops) is pathognomonic.

Knee-jerk never wanting.

Leukocytosis absent.

Choroid tubercles absent.

Cultures from punctured spleen may show typhoid-bacilli (dangerous procedure).

Hemorrhage from the bowels common.

Perforative peritonitis often present.

## PULMONARY FORM.

Though all gradations between the typhoid and the pulmonary types occur, the latter should be recognized and briefly described. It may develop suddenly, the ushering-in symptom being sometimes a chill, though more frequently there is a premonitory period, during which the general health fails materially. The affection may follow promptly upon some acute illness, such as measles or whooping-cough, in children, in which there has been marked catarrhal bronchitis.

*The respiratory symptoms* are early prominent, and later preponderate in the clinical picture. From the start there is dyspnea, and this gradually increases until the respirations become rapid (40 to 60 per minute). When dyspnea becomes pronounced the face assumes a characteristic cyanotic pallor. The cough, at first, is moderately severe, but soon it becomes troublesome, being now frequent and attended with a slight expectoration, which is non-characteristic.

*The physical signs* are those of broncho-pneumonia, and the latter may or may not be preceded by the signs of general bronchitis. With the onset of broncho-pneumonia there appear spots that yield either dulness or a tympanitic resonance on percussion, and broncho-vesicular breathing with numerous subcrepitant râles on auscultation.

*The general symptoms* are marked from the beginning. The fever is high—from 103° to 105° F. (39.4°–40.5° C.) or often higher. The pulse ranges from 100 to 140, is small, feeble, and sometimes irregular, and it may be more rapid still during the advanced stage of the affection (see Fig. 25). Cerebral symptoms rarely appear.

<sup>1</sup> See also Differential Diagnosis of Typhoid Fever.



The **course**, as a rule, is more prolonged than that of general miliary tuberculosis, except in children, in whom it often runs an exceedingly acute course. As the end approaches the signs of suffocation are gradually intensified, and finally lead to a fatal termination.

**Diagnosis.**—The diagnosis is difficult; but a family history of tuberculosis, a knowledge of the pre-existence of a tuberculous focus or of an antecedent predisposing affection, will aid in its recognition. Tubercle bacilli are perhaps not demonstrable in the sputum unless an old tuberculous lesion coexist. In doubtful instances; however, an attempt should be made to detect the bacilli in the blood. Occasionally either tuberculous meningitis or peritonitis supervenes, and aids in removing the doubt, and in a small percentage of the cases choroid tubercles are detectable. These points, together with the more marked general symptoms, will usually aid the clinician in distinguishing this variety of tuberculosis from non-tuberculous broncho-pneumonia.

#### CEREBRAL OR MENINGEAL FORM (TUBERCULOUS MENINGITIS).

This variety is of quite frequent occurrence, appearing in not less than 50 per cent. of the cases of miliary tuberculosis. When it develops the symptoms referable to other organs than the meninges are in abeyance. With reference to the etiology of this form one fact needs to be emphasized—namely, that most cases are observed between the ages of two and seven years; it may, however, be met with at any time of life. The affection frequently has its origin in tuberculous bronchial glands (Jacobi), and the history of a fall is common. A few cases have been found to be associated with erythema nodosum. Exceptionally the meninges are primarily involved.

**Pathology.**—The chief site of the tubercles in children is the pia mater at the base of the cerebrum (basilar meningitis), whilst in adults the pia at the vertex is more apt to be involved. The membrane surrounding the tubercles may not be inflamed, there being a simple tuberculous deposit. On the other hand, more or less inflammation, with sero-fibrinous or fibrino-purulent exudation, is generally present in the region of the base. This exudate is usually abundant in the Sylvian fissures, and may find its way to the external surface of the hemispheres. It is gray in color, transparent, and gelatinous, and contains in its meshes the tubercles, which appear as grayish-white bodies, and which, in cases of equal severity, may be either numerous or scanty. They may be scarcely visible to the naked eye, but may vary from the size of a pinhead to that of a French pea. The branches of the Sylvian artery may be implicated, either owing to the direct pressure of the exudate or to the obliterating arteritis produced by a tuberculous infiltration. The pia looks like wet blotting-paper over the quadrangle at the base (Gray). Elsewhere it is thickened and opaque, though easily detachable. Osler says: "The arteries of the interior and posterior perforated spaces should be carefully withdrawn and searched, as upon them nodular tubercles may be found when not present elsewhere. In doubtful cases the middle cerebral arteries should be very carefully removed, spread on a glass plate with a black background, and examined with a low objective. The tubercles are then seen as nodular enlarge-

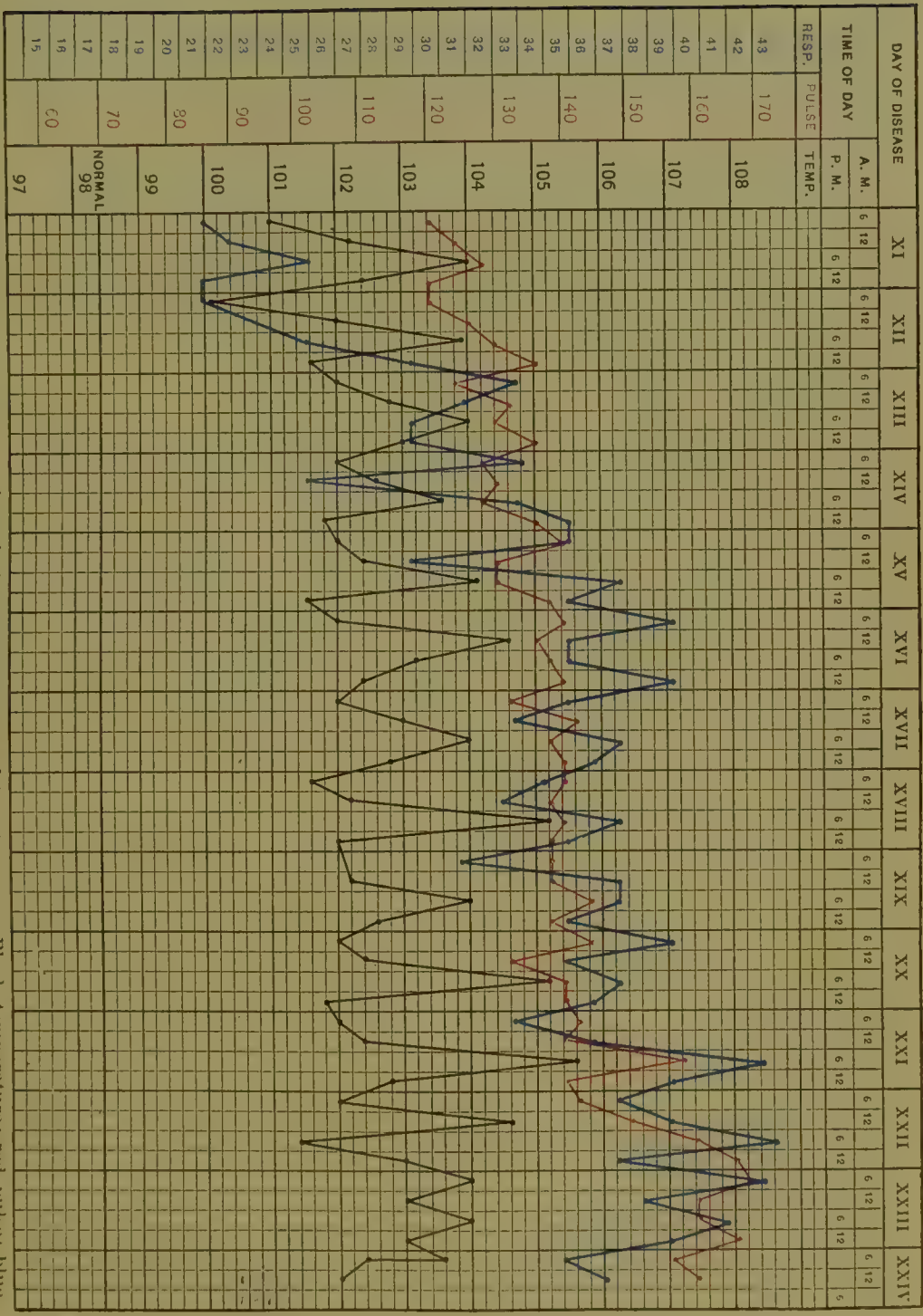


Fig. 25.—Chart of a case of acute pulmonary tuberculosis. Mrs. M—, aged twenty years. Black, temperature; red, pulse; blue, respirations.

ments on the smaller arteries." Involvement of the chief vessels that nourish the walls of the ventricles and the ependyma, and stretch from the vermis cerebelli forward over the quadrigemina, explains the constant presence of a turbid fluid in the ventricles, with softening of their walls. As the result of undue intraventricular pressure the cerebral convolutions become more or less flattened, with effacement of the sulci. The cortex, to a variable depth, is generally the seat of red softening, and more rarely of white softening alone. The tuberculous infiltration involves the cranial nerves.

*Histology.*—The tubercles grow in the perivascular sheaths, which are often distended with lymphoid and epithelioid cells, and there is observed not infrequently a thrombosis of the arteries and of the venules of the pia, obliterating their lumen. The pia mater is gradually thickened through cellular infiltration, and in a small proportion of the cases the spinal meninges are similarly involved, chiefly in the cervical portion of the cord.

**Symptoms.**—There is a prodromal period which lasts one or more weeks, during which the patient (usually a child) is pale, peevish, has headache and photophobia, and grinds its teeth during sleep; the tongue is coated, appetite impaired, and there may be occasional vomiting, either propulsive or regurgitative. Constipation is present and may be marked. Among rare premonitory symptoms are slight hyperesthesia of the abdomen and a diminished urinary secretion. A tendency to emaciation is quite constant. These prodromal symptoms present variations as to their number and combinations in different cases. In few instances only is the onset acute. The symptoms usually indicate basic meningitis, and at first there is associated considerable mental excitement; later there are pressure-symptoms (caused by the exudate), with total loss of the mental faculties.

(1) **Stage of Cerebral Excitement.**—The *invasion* is generally gradual, or even quite insidious, its most characteristic phenomena being *severe vomiting, marked headache, and chills followed by fever*. Certain other symptoms now arrest the attention, such as extreme irritability, screaming, and great obstinacy, and occasionally drowsiness appears early. When the onset is sudden the disease may be disclosed by convulsions, paralysis, wild delirium, or coma. The established disease exhibits certain distinctive features. The pain is often most excruciating, causing the child to utter short penetrating screams (hydrocephalic cry), and in rare instances the sharp cries may be continuous and lead to physical exhaustion. The headache is increased by light, noise, or movement. Vertigo is common; the pupils are contracted at this period; the face pales and then flushes; the pupils alternately dilate and expand; and the expression is sometimes sad, though more often stupid. Generally hyperesthesia or dysesthesia may appear, and there may be a slight mind-wandering at night, though active delirium is rare. *Tâches cérébrales* may be obtained, but are not characteristic. The patient is intolerant of every form of disturbance. All the symptoms of the prodromal stage are now aggravated; slight muscular twitchings and sleep-starts occur; the vomiting is apparently causeless, and may be frequently repeated; and constipation persists.

Fever is present, but is of slow development, and rarely rises higher



than 102° or 103° F. (39.4° C.) in the evening. The skin is dry and harsh, as a rule. The pulse is slow or moderately accelerated, but soon quickens to 120 or even 130, and later it may be irregular. At times the pupils are unequally contracted, and ptosis may also be looked upon as an early sign.

(2) **Second or Transitional Stage.**—The symptoms of cerebral irritation now abate, the patient becoming more quiet, while mental dulness often supervenes. The vomiting and headache subside gradually, and the child cries out only occasionally. The abdomen is now distinctly scaphoid and the head occasionally retracted. Constipation is obstinate. The evidences of localized organic foci, such as slight twitchings of the muscles of the face, followed by strabismus, ptosis, or paralyses of the face or limbs, may appear. Generalized convulsions may occur, and muscular tremors and athetoid movements may appear. Both pupils (or one only) may be dilated as intracranial pressure develops; patchy flushing of the face is common. The respiration is now irregular and sighing.

(3) **The Stage of Paralysis.**—On account of the exudation the mental faculties are abolished, so that the patient is comatose, though convulsions or localized spasms of the muscles in different parts of the body (neck, back, limbs, etc.) may be observed. Optic neuritis develops, while the paralysis of the ocular muscles above noted deepens. The pupils are dilated, the eyes are partly closed, and the eyeballs at intervals slowly and alternately move in a lateral direction. Hemiplegia sometimes develops, and more rarely monoplegia, affecting the face or one of the extremities. There may be paralysis of the third nerve, with involvement of the face, hypoglossal nerve, and limbs on the opposite side (a combination of symptoms first observed by Weber), consequent upon a lesion localized in the internal inferior portion of the crus. Monoplegia of the right side of the face has been observed in a few instances, associated with aphasia. Exceptionally aphasia and brachial monoplegia have been combined. The temperature in the early part of this stage usually rises to 103° F. (39.4° C.) or higher, but later it may drop to a subnormal level, and in rare instances as low as 94° F. (34.4° C.). Immediately preceding the fatal termination the temperature may rise to 106° or 107° F. (41.6° C.), the pulse becoming frequent, small, and irregular. Gradual anesthesia comes on with general muscular relaxation.

Occasionally a typhoid state (great prostration, dry tongue, diarrhea, etc.) may develop, and Cheyne-Stokes respiration is almost invariably present, preceding the fatal event. Leukocytosis has been observed in all stages of the disease.

*Ophthalmoscopic Examination.*—The ophthalmoscopic appearances sometimes form important points in the diagnosis. At first hyperemia of the disk is noted, and later the changes belonging to neuritis (swelling and striation) appear, while rarely tubercles may be detected in the choroid.<sup>1</sup>

**Clinical Types.**—(a) **Mild Type.**—The marked or alarming symptoms (tetanic rigidity of the muscles, convulsions, and paralysis) develop at a late period. In this class should be placed those cases in which the

<sup>1</sup> The differential diagnosis is given in the section on Meningitis.

meningitis is but feebly indicated—*e. g.* when it is but a small factor in the condition of acute general tuberculosis.

(*b*) **Malignant or Rapid Form.**—This type is comparatively rare, occurring most frequently in adult life, while the lesions have their seat almost exclusively upon the convexity. The onset is marked by the most frightful tetanic convulsions, which precipitate a fatal termination in a couple of days.

(*c*) **Chronic Type.**—Cases pursuing a chronic course are rarely encountered, and the symptoms usually point to localized cerebral lesions (Jacksonian epilepsy, etc.).

**Prognosis.**—The disease lasts from two to four or five weeks, though chronic cases may continue for several months. When the convexity is implicated, however, the duration is only one or two weeks. It should be emphasized that frequently in the course of well-marked cases a decided remission in the leading symptoms occurs, so that convalescence is suggested; but this is deceptive, and is almost invariably followed by a renewal of the unfavorable features of the affection. A few cases only are recorded in medical literature as ending in recovery.

Freyhan has reported a case with recovery in which the diagnosis was proved by puncture of the spinal canal and the withdrawal of fluid, in the sediments of which tubercle bacilli were found. A. Jacobi has met with 2 cases that terminated favorably, and Leube has also reported a case in which the symptoms were characteristic, and at the autopsy, some years later, old tuberculous lesions were found in the meninges. It is to be recollected, however, that the course of tuberculous meningitis is probably uninfluenced by human agency.

### ACUTE PNEUMONIC PHTHISIS.

(*Acute Phthisis; Florid Phthisis; Galloping Consumption.*)

This may be primary or secondary, the latter form being consequent either upon a localized tuberculous area in the lung, tuberculous pleurisy (acute or chronic), tuberculous peritonitis, or tuberculous disease of some other organ. Acute phthisis may occur at any age, though it is relatively more frequent in childhood and early adult life, but whether primary or secondary, the infection of the lungs is rapid.

**Pathology.**—Two forms may be recognized: (1) This reveals the appearances of an *acute lobar pneumonia*, one lobe only being implicated, as a rule, though sometimes the whole lung is involved. The process leads to a destruction of lung-tissue, so that a section may show the existence of cavities. The latter are usually small, while surrounding them may be seen tubercles in hepatized tissue, and here and there caseous masses of a yellowish-white color may be visible. These often indicate old or pre-existing foci. It is sometimes exceedingly difficult to distinguish a tuberculous croupous pneumonia from the ordinary form, and the most careful inspection may fail to reveal the presence of elementary tubercles in the acutely consolidated tissue. In cases in which this disease is suspected, however, the opposite lung, the bronchial glands, and the peritoneal and other organs should be carefully examined.

The lesions presented by cases that have run a long course are somewhat characteristic, though not always the same. If the case has



had a duration of eight or ten weeks, apical softening with more or less extensive cavity-formation often occurs. Less frequently, a lobe or an entire lung is found to be consolidated throughout, "and converted into a dry, yellowish-white, cheesy substance, in which condition it may remain till the end."

(2) *Presenting the Appearances of Broncho-pneumonia.*—This variety is more common than the previous, especially in children. The evidences of bronchitis affecting the finer tubes, together with consolidation of the lobules to which the tubes lead, are striking. As in ordinary broncho-pneumonia, so here, the solidified areas appear as grayish-red masses in the early stage, while later they are of an opaque-white. The products that fill the air-cells may caseate and break down, with the formation of irregular cavities that vary in size. When large areas are involved they are the result of the fusion of contiguous smaller areas of hepatized tissue. The trouble often begins in the upper lobes and spreads downward, though not infrequently the lower lobes are most extensively involved.

In not a few cases the masses are small, multiple, and widely disseminated throughout the lungs, and miliary tubercles in the lungs or pleuræ are associated with the broncho-pneumonic lesions before described. In nearly all cases signs of pleurisy may be noted, as is shown by pleural adhesions or by deposits of lymph on the pleura. The bronchial glands are also usually infected, and, particularly in children, are the seat of tuberculous processes.

Bäumler has called attention to a type of *tuberculous inhalation pneumonia* consequent upon hemoptysis, the blood and contents of the cavities being drawn into the finer tubes in respiration. This form of broncho-pneumonic phthisis sometimes follows pulmonary tuberculosis in the early, though more often in its late, stage. On microscopic examination tubercle bacilli are found, though rarely in abundance, in the infiltrated masses and in the walls of the cavities.

**Clinical History.**—(1) **Acute Cases.**—Preceding the attack, the patient may have "taken cold" or have been in a run-down state; more often, however, he has been apparently healthy. The onset is sudden, marked by more or less rigor, pain in the side, fever, cough, and great prostration, and there may be bronchial hemorrhage which may last one or more days. The total amount of blood expectorated may be considerable. In the majority of cases the expectoration is mucoid at first, and then becomes rusty-colored, often containing tubercle bacilli, though at first they may be absent, and indeed not appear until late in the disease. Dyspnea appears early, and may soon become extreme, and the fever quickly rises to 104° F. (40° C.) or over. It may be of the continued type or it may early assume the remittent or hectic type, and with the latter forms of fever, which usually begin about the end of the first week, are associated night-sweats and rapid emaciation. The prostration of the vital powers is now extreme. The expectoration is more abundant, muco-purulent, and often greenish-yellow in color.

In the course of one or two days after the onset we obtain physical signs that vary with the extent of the lesions. Usually, as before stated, these cases present the anatomic appearances of acute lobar pneumonia—viz. the complete consolidation of one or more lobes, which



is usually followed by signs of softening, provided the patient survives the first week or ten days. The physical signs during the stage of consolidation are precisely the same as in lobar pneumonia. The signs of softening and of cavity will be given in detail in the description of Chronic Phthisis.

The *course* is usually rapid, occupying from two to six weeks on the average, though rarely cases that reach the stage of cavity-formation are protracted to three or even four months. Considering the brevity of the attacks, the extreme degree of emaciation (shown especially by the hollow cheeks and temples, pinched nose, and thin hands) is truly remarkable. The patient usually maintains a hopeful state of mind, notwithstanding the rapid downward course of the affection, and it may be admitted that recovery is possible. The parts involved are in such cases destroyed and replaced by fibrous tissue, and it should be remembered that the apex is involved in most cases. It sometimes happens that consolidation only is present in the second lobe affected, while in the upper lobe one or more cavities have already been developed. The pleural crepitating friction is often audible before consolidation is complete.

*Diagnosis.*—The onset, symptoms, and course during the first week may be those of ordinary lobar pneumonia, but in some cases certain symptoms may arise which will excite suspicion of their tuberculous character in the early stage. Thus, hemoptysis rarely occurs in a pneumonia due to pneumococcus infection, and, what may also serve as a point in diagnosis, the appearance of the patient, as well as his previous and family history, may be suggestive. The points of discrimination have been fully set forth in the section on Lobar Pneumonia (pp. 150, 151).

(2) **Subacute Cases** (rarely acute).—The onset is less sudden than in the former type, while the patient's antecedent condition may either be good or below the *standard*. At the beginning he has repeated chills, though hemoptysis may be the first symptom which indicates a pre-existing tuberculous focus. The fever rises high, and is apt to be irregular from the start; the pulse and respirations are rapid, and there is a muco-purulent expectoration which may either be profuse or scanty. Occasionally it is fetid, and the sputa may early contain elastic fibers and tubercle bacilli, though more often these are noted after the affection has become fully established. During the progress of the case, also, hemoptysis may arise. Later, drenching night-sweats increase the exhaustion and emaciation, which speedily reach an extreme degree, and soon or late a typhoid condition of the system is developed.

The *physical signs* are, at first, those of general bronchitis, with or without indications of pleurisy. Later, small areas of consolidation, which often increase in size, are indicated by impaired percussion resonance or dulness and by broncho-vesicular (rarely tubular) breathing, with subcrepitant râles. These signs may be unilateral, though more often they occur bilaterally. In many cases softening with cavity-formation ensues, with the usual physical signs of this condition.

*Course and Duration.*—For some time the patient may remain out of bed, though in most instances the disease constantly progresses. Less frequently there are exacerbations and remissions. A small

percentage of these cases recover with a loss of more or less lung-tissue, though the condition may pass into chronic phthisis. It is important to recollect that the local lesions may become extensive, as the result of fusion of small consolidated masses, until an entire lobe is involved, and when this occurs the symptoms and course simulate those of the acute type. The duration ranges from two to eight weeks or more.

*Diagnosis.*—This variety is frequently confounded with non-tuberculous broncho-pneumonia, and the chief distinctions will be mentioned in connection with the latter disease. *Bronchiectasis* may be accompanied by emaciation, fetid expectoration, night-sweats, and the signs of cavity, and these cases have been mistaken for acute phthisis. Important in the recognition of the latter, however, are marked fever and emaciation. Moreover, the physical signs are more frequently referable to the apices, and the disease is more steadily progressive, running a shorter course than bronchiectasis.

**Acute Broncho-pneumonic Phthisis in Children.**—The belief that the form of broncho-pneumonia that so frequently follows certain infectious diseases (measles, whooping-cough, etc.) is in the majority of instances tuberculous has been steadily gaining. Osler recognizes three groups of cases: (*a*) Those in which the child suddenly becomes ill while teething or during convalescence from fever, with high temperature, severe cough, and the signs of consolidation of one or both apices. Death may occur within a few days. To the naked eye the lesions do not appear to be tuberculous. (*b*) In this group the children show the ordinary symptoms of broncho-pneumonia, and the cases are more protracted, death occurring about the sixth week. (*c*) The child feels ill during convalescence from an infectious disease, fever, cough, and dyspnea being present. The intensity of the symptoms abates within a fortnight, and the physical examination shows the presence of diffuse bronchitis with scattered minute areas of consolidation. Many of these cases develop into chronic phthisis.

## CHRONIC TUBERCULOSIS.

(*Chronic Pulmonary Tuberculosis; Chronic Ulcerative Phthisis.*)

This form is much more common than the acute, the term embracing sub-varieties to which attention will be incidentally directed. Its most typical clinical form follows a mixed infection as a result of a septic element superadded at some time to the primary tuberculous infection.

**The Causal Factors** have been detailed under General Etiology.

**Pathology.**—The pathologic characters of tuberculosis in general have been already presented, but it will be necessary to describe briefly the special anatomic conditions met with in chronic ulcerative phthisis.

The post-mortem appearances of the lungs in chronic pulmonary tuberculosis are remarkable for their great diversity, not only in the extent of tissue involved, but also as to the character of the morbid processes. Often the associated lesions form no unimportant part of the picture. In nearly all fatal cases the most advanced and extensive lesions are found near the apex, and, as a rule, the entire upper lobe of one of the lungs is implicated. In addition, it is observed that the destructive process has extended to the lower lobe of the same side as



well as to the apex of the opposite lung, the lower lobe of the primary lung generally being invaded before the upper part of the other. Though both lungs are affected in fatal cases, they represent different stages of the disease. The case is very different in an old and cured tuberculosis of the lungs, such as is frequently met with in persons who have died of some other affection. Here the lesions may occupy but a small part of one lung, and usually near the summit.

Kingston Fowler has investigated the question of the points of election and paths of distribution of the lesions in chronic phthisis, and has found that the primary lesion is not, as a rule, at the summit of the upper lobe, but that it occurs from 1 to  $1\frac{1}{2}$  inches (3.79 cm.) below this point and near the postero-external borders. Favored by normal respiration, the lesions advance downward, so that on physical examination the first evidences of disease are to be found posteriorly over the lower part of the supraspinous fossa, while anteriorly the early signs are met with immediately below the middle of the clavicle, extending along a line running about  $1\frac{1}{2}$  inches (3.79 cm.) from the inner end of the second and third interspaces. The starting-point, though less frequently, may also be indicated by physical signs in the first and second interspaces below the outer third of the clavicle, with subsequent downward extension.

From personal observation of the post-mortem lesions of this disease, and from my studies at the bedside, I feel convinced that the initial lesion is frequently located anteriorly and near the apex, corresponding on the chest-walls to the clavicle and the supraclavicular spaces. This site has seemed to me to obtain more often on the right side than on the left. Kingsley has shown that when the lower lobe becomes involved the consolidation begins about  $1\frac{1}{2}$  inches (3.79 cm.) below its apex posteriorly, and corresponding externally to a spot opposite the fifth dorsal spine. From this point it spreads downward and laterally in a line following the border of the scapula "when the hand is placed on the opposite scapula and the elbow rests above the level of the shoulder." The middle lobe on the right side is usually invaded by direct extension from the upper. The seat of primary infiltration may even be the lower lobe, but this is an occurrence of great rarity.

The relative frequency of involvement of the two sides varies according to different authorities. A careful analysis of my own records, and the results of some statistical investigations into the subject, show that out of a total of 1236 cases 726 occurred on the left side and 510 on the right.

In all cases the primary lesions are due to tuberculous infiltration, which at first is confined to certain lobules, though it may later involve extensive areas of lung-tissue (tuberculous broncho-pneumonia). In most instances the starting-point of the morbid changes is in the smaller bronchi and also, according to Payne, the inside of the alveoli. Soon the bronchioles and the corresponding air-cells become blocked with inflammatory products. These areas then undergo caseation and present the usual opaque, grayish-yellow appearance, a cross-section of these yellow nodules showing the central bronchus usually plugged with exudate and surrounded by caseous matter. Softening and sometimes complete liquefaction, with expectoration or absorption of the altered



morbid products, may take place, and this disintegration is associated with *ulceration* in the wall of the bronchus, consequent upon secondary pyogenic infection, and a resulting formation of small *cavities*. Ulcers may form in the bronchioles before necrotic processes supervene, and they are generally shallow, with sharply-defined edges. Recovery may ensue as the result of *calcification* with encapsulation of the cheesy masses, or the affected area may undergo *fibroid transformation*—a conservative process and one that may lead to actual cure. It often happens, however, that old and apparently healed tuberculous lesions take on ulceration, when the calcareous masses (pulmonary calculi) may be dislodged and expectorated, and the more rapidly the caseous masses are formed the more liable are they to softening. Surrounding the healed areas the tissue may be the seat of atelectasis, though more often of emphysema. Destruction of lung-tissue also results from interstitial inflammation with the formation of new connective tissue, the latter in turn compressing and finally obliterating the alveoli.

**Cavities** (*Vomicæ*).—These result chiefly from progressive necrosis and ulceration. They are formed mostly by dilatation of the bronchi, whose walls are tuberculous and suppurating. But they may also arise independently of the bronchi. Cavities vary largely in number, size, form, and in other characteristics. They are often multiple, though usually not far removed from one another, and unite as they increase in size. In this way large cavities, involving the whole of one lobe and even an entire lung (except the extreme anterior margin), may be formed, and a variable number of small pockets connecting with the bronchus may thus originate. The walls of the cavities are almost invariably irregular.

*Vomicæ* may be classified as (1) progressive and (2) non-progressive.

(1) The **progressive** are divisible into (a) New cavities and (b) Old cavities.

(a) *New cavities* have soft, necrotic, friable walls so long as the destructive processes are rapidly progressing, and the same state of things prevails in the cavities of acute phthisis. They may develop near a healed focus or near old cavities with limiting walls, and when situated near the periphery of the lung they may rupture into the pleura, causing pneumothorax.

(b) *Old cavities*, as a rule, have sharply-defined walls that vary considerably in thickness. At first they consist of a fibro-vascular zone, which has an inner suppurating surface; subsequently the lining of this zone is converted into an exfoliating membrane. The contents of *vomicæ* are muco-purulent or purulent, and often consist of a shreddy and sometimes a bloody fluid. Rarely they are gangrenous. Cavities also contain tubercle bacilli and other micro-organisms. Percy Kidd has studied the question of the relation of tubercle bacilli to tuberculous pulmonary lesions, and states that they are invariably present in newly-developed tubercles and fresh cavities, but frequently absent in old nodules. Trabeculæ composed of blood-vessels and remnants of pulmonary tissue often traverse the cavities. In old cavities excavation may be complete, not a vestige of normal or diseased tissue remaining in them, though the blood-vessels, many of which are beaded by small aneurysmal dilatations along their course, are the last to disappear.

Their removal is effected by an obliterating inflammation. Rupture of these miliary aneurysms or the erosion of a large vessel is an event that gives rise to copious hemoptysis. Cavities having dense walls may also increase in size by encroaching upon and destroying the surrounding tissue, huge cavities often having thin, tense walls. But, wherever situated, they usually begin toward the summit of the upper lobe. Another common seat is the mid-dorsal region.

**Non-progressive Cavities.**—Quiescent cavities are usually small, though variable in size, according to the stage at which the process of contraction is arrested. Medium-sized and large vomicæ do not become totally occluded. They may be multiple, though more often perhaps single, and associated with them may be observed dense, fibrous nodules representing healed foci. Their interior may be lined with a smooth, cuticular structure resembling mucous membrane.

**Interstitial Pneumonia.**—In the course of chronic phthisis interstitial inflammation of two sorts will most probably arise: (a) A consolidation excited by the tubercle bacilli themselves, and hence manifesting a *destructive* tendency; (b) A slowly-developed *interstitial pneumonia* which aims at *arresting* the progress of the affection. It develops in close proximity to caseous masses and around cavities. The new connective tissue thus formed in obedience to the well-known pathologic law tends to contract secondarily, and thus vomicæ are often partly, though seldom entirely, obliterated. The shrinking of the connective tissue may also result in compression, and finally in the destruction of pulmonary tissue, just as in a tuberculous inflammation. The process in this instance, however, is on the whole conservative and calculated to repair tuberculous lesions.

**Disseminated Tuberculosis.**—*Miliary Tubercles.*—This form has for its chief characteristic miliary tubercles, which are scattered not only about the tuberculous area, but also throughout the rest of the lung, and usually in the lower lobe. Most of the tubercles undergo fibroid or fibro-caseous change. These minute, hard gray or grayish-yellow nodules vary in size from a mustard-seed to that of a pea, and lung-tissue that is more or less studded with chronic miliary tubercles is apt to look pale, while the surrounding air-cells are *emphysematous*. The condition may lead to pneumonia, and the whole aspect then becomes altered. Here, as before described, fusion of miliary tubercles results in larger masses which become caseous, and hence the method of cavity-formation is identical with that observed in tuberculous broncho-pneumonia. In the disseminated form tubercles may also be found in many other organs than those indicated (pleura, trachea, larynx, bronchial and other lymphatic glands, peritoneum, spleen, kidneys, liver, brain, mucosa, testes, etc.).

**Lesions of the Pleura.**—This membrane is hyperemic and coated with fibrinous exudation coextensively with the affection of the parts in chronic ulcerative phthisis. The pleural membranes are only more or less thickened by organized adhesions, but in the latter and also in the pleura tubercles or cheesy masses may be found. Simple and other forms of pleurisy are also met with—sero-fibrinous, purulent, and hemorrhagic.

**Lesions of the Bronchial Glands.**—At first these are enlarged and



edematous, containing tubercles, and later they present foci which often undergo purulent disintegration and sometimes calcification. Other lymphatic glands than these may be affected (mesenteric, etc.).

**Lesions of the Larynx.**—The larynx is frequently the seat of tuberculous infiltration and ulceration, particularly in certain parts, such as the vocal cords, posterior wall, ary-epiglottidean folds, etc.

**Lesions of the Heart.**—Tuberculous endocarditis is present in about 5 per cent. of the cases, and congenital stenosis of the pulmonary orifice is noted in not a few instances (Chevers). The right heart is often hypertrophied or dilated.

*Other organs* may present lesions in chronic phthisis, and these will be spoken of in connection with the clinical history.

Tuberculosis of the intestinal canal is a common though late lesion.

*Amyloid degeneration* of certain organs is a not unusual secondary event, especially of the kidneys, liver, spleen, and intestinal mucosa, and in like manner enlargement of the liver due to *fatty infiltration* is noted not infrequently.

**Clinical History.**—The mode of invasion is quite diverse, but with few exceptions the onset is either (1) gradual or (2) abrupt, and, as a rule, the health has been previously undermined for a longer or shorter period.

(1) **Gradual Onset.**—(a) Most frequently the disease originates in a manner similar to the origin of *ordinary bronchitis*, and often, combined with the symptoms of broncho-catarrh, are those of pleurisy. Tuberculous bronchial affections often follow certain acute infectious diseases—influenza, typhoid, measles, whooping-cough, etc.—and in this form are rarely curable. The physical signs may be negative for some time, and then appear at the apex of the lung. Over a small area there may now be slightly impaired resonance on percussion, with harsh broncho-vesicular breath-sounds and with or without subcrepitant râles. The expansion, as noted on inspection and palpation, over the affected spots is more or less defective, while the vocal resonance and fremitus are either increased or unaltered; and the fact that the lesions are more commonly detectable in the suprascapular fossa than anteriorly must be remembered. At this period obvious constitutional disturbances are present (debility, emaciation, fever, etc.).

(b) *Onset with Pleurisy.*—This may be sudden, as in an acute pleurisy with effusion, but often the latter condition develops insidiously. Of 90 cases of pleurisy with effusion, one-third terminated in chronic phthisis (Bowditch). It may begin as a dry pleurisy at the apex, either anteriorly or posteriorly, or the evidence of pleurisy may be associated with the more common or bronchitic onset.

(c) *With Gastro-intestinal Symptoms.*—There is impaired digestion, and soon the patient becomes anemic, loses flesh, and is debilitated. Later, the first indications of pulmonary tuberculosis develop in the lungs.

(d) *With indefinite peritoneal symptoms*, lasting for months or even years.

(e) *With Laryngeal Symptoms.*—This is a rare form. It begins with hoarseness, more or less aphonia, and considerable cough; there is also a slight muco-purulent expectoration. Laryngoscopic examinations may



detect tuberculosis of the organ, and tubercle bacilli may be found in the sputum before involvement of the lungs is discoverable.

(2) **Cases with Abrupt Onset.**—(a) The most important group under this category is heralded by the symptoms and signs of *acute pneumonia*, more commonly of the lobular variety. As compared with ordinary pneumonias, these present some peculiar features: the fever is irregular and the expectoration is more abundant, is blood-stained, and contains bacilli. The signs are usually located in the apical region. Resolution may occur, but recovery is not complete, and the condition is likely to pass into chronic phthisis.

(b) *Onset with Fever.*—Chills and fever are apt to arise in all instances in the advanced stage of pulmonary tuberculosis, and these symptoms may also initiate the attack. There is no mistake in diagnosis more commonly made in malarial regions than to ascribe such cases to paludism.

(c) *With Hemoptysis.*—This symptom may be the first to invite attention to lung-trouble. In the majority of cases the amount of blood lost is considerable, and, less frequently, repeated slight hemorrhages occur. Pulmonary symptoms may be absent, sometimes temporarily, and in rare instances, perhaps, permanently; but in a great proportion of cases the clinical picture of incipient pulmonary tuberculosis is revealed pursuing its accustomed course immediately after the occurrence of the hemorrhage. The physical signs may be latent for a time, and, whilst they are usually found to be at the apex, they may assume the guise of a pleurisy in the scapular or infrascapular region. A slight tuberculous lesion is most probably present in these cases before the occurrence of the hemorrhage.

The **symptoms** are (1) *local* and (2) *general*.

(1) **Local.**—(a) *Pain.*—This is absent in many cases of chronic phthisis and in others it may be moderately severe. It is seated usually at the base, laterally or anteriorly, and not rarely there is pain of a lancinating character in the interscapular region in the early stages of the affection. This symptom is of diagnostic worth only after other forms of pain (rheumatic, neuralgic, etc.) have been excluded. The most common cause of pain is pleuritis, with or without pleuritic adhesions; it is increased on deep breathing and coughing. Intercostal neuralgia and pleurodynic stitches may also develop in the course of this disease.

(b) *The Cough.*—This may be looked upon as an essential feature, though in a few instances it may be slight or even wanting throughout. Its severity bears no constant relation to the extent of the pulmonary lesions, but rather to the degree of sensitiveness of the patient. It is dry and hacking at the beginning, and, if the larynx be involved, the cough is marked and takes on a hoarse quality. It is most pronounced at certain periods of the day—viz. on lying down at night and on awaking from sleep. Paroxysms may also occur after meals, and these occasionally induce vomiting. The cough is at times distressing and debilitating in its effects.

(c) *Expectoration.*—At the beginning the sputum is scanty and mucoid, rarely hemorrhagic, or it may be merely streaked with blood; later it may become muco-purulent, and the appearance of small gray

or grayish-yellow flocculi first suggests the nature of the affection. With the onset of the stage of cavity-formation the sputum becomes more abundant and more distinctly purulent, and, after the formation of cavities of any size, airless, opaque, and nummular (coin-shaped) masses are expectorated. The latter are greenish-gray or greenish-yellow in color, and sink rapidly when discharged into water. They are often mingled with more or less bronchial secretion, and are not entirely characteristic of tuberculous cavities, being sometimes observed in pure bronchitis. They may even be absent, and the expectoration be merely purulent. The opening of a fresh cavity may be followed by very free expectoration. The sputum is sometimes fetid, and exceptionally it is horribly offensive, varying greatly in amount in different cases and at different stages of the disease. In certain cases it is absent throughout the greater portion of their course, and is especially apt to be slight in children and old people. In such instances it may be impossible to collect sufficient sputum to examine for bacilli.

Microscopic examination discovers alveolar epithelium (particularly in the earlier stages), pus-cells, blood, fat-globules, elastic fibers, and tubercle bacilli, the detection of the latter being the most important factor in the diagnosis. It may be safely stated that the finding of bacilli in the sputum is *prima facie* evidence of chronic phthisis; on the other hand, however, their absence in the early stage does not exclude the disease. It is often needful to make repeated and delicate examinations of the sputa—a course that will finally bring ample reward if the case is one of phthisis. It is also of the utmost importance to select for examination the small grayish masses that are usually to be found, since they early contain the bacilli.

“A small amount of the purulent portion of the sputum is spread in a thin and uniform layer on a perfectly clear cover-glass by means of forceps, needles, or the Ohse, which must previously be held a moment in the flame of a Bunsen burner or a spirit lamp, or by pressing a small amount of sputum between two cover-glasses, then sliding them apart. It is then dried in the air, or more quickly by holding the cover-glass with forceps some distance above the flame of a burner or lamp. Finally, it is to be passed three or four times through the flame, and so ‘fixed’” (Musser).

The preparation may be stained with carbol fuchsin (basic fuchsin 1, alcohol 10, 5 per cent. solution of carbolic acid 90), either by dropping a few drops of the stain on the smeared side of the cover-glass and holding it above the flame until it steams, or by floating its face downward upon a watch-crystal containing the solution. It must then be decolorized either with a 10 per cent. solution of nitric acid, allowing it to remain until the red color has entirely disappeared (about fifteen seconds), and then washing and counter-staining with methylene-blue, or with Gabbett's solution (methylene-blue 2 gm., sulphuric acid 25 c.cm., water 75 c.cm.), in which it must remain until the red color has been replaced by a faint blue (thirty seconds or more). Instead of carbol-fuchsin, anilin gentian violet may be employed (add a saturated alcoholic solution of gentian violet to a filtered saturated solution of anilin until a metallic luster appears on the surface). The specimen may lie either several hours in a cold solution or a few minutes in one

that is steaming. Decolorize with the nitric-acid solution and counter-stain with rubin or Bismarck brown. It is often much simpler to smear the sputum directly upon the slide, and then examine, when stained, without the intervention of a cover-glass. A much larger amount of sputum can thus be prepared at a single operation.

In the microscopic examination use a  $\frac{1}{2}$ -inch (2.11 mm.) oil-immersion lens and Abbe condenser, or, at the least,  $\frac{1}{4}$ - or  $\frac{1}{8}$ -inch (0.36 cm. or 0.31 cm.) objective. If carbol-fuchsin has been used in staining for the bacilli, and methylene-blue as a contrast, the former will be found as red rods in a blue field (background), while if gentian violet has been used, the tubercle bacilli appear as dark blue rods, with all other bodies brown, if Bismarck brown is used for the contrast stain. There may be visible in the field a few bacilli only, particularly during the early part of the case. In this stage of cavity their number is usually increased, and sometimes they are quite numerous.

The demonstration of *elastic fibers* is also an important aid to diagnosis. Fenwick's method is the following: Boil the sputum with an equal quantity of a solution of caustic soda (gr. xv-3j—0.972-32.0); pour the product into a conical glass and fill with cold water. The sediment is subsequently examined with care for elastic fibers.

The method of Sir Andrew Clark possesses the advantages of being simple and speedy: "The thick, purulent portions are placed on a glass plate, 15 × 15 cm., and flattened into a thin layer by a second glass plate, 10 × 10 cm. In this compressed grayish layer between the glass slips any fragments of elastic tissue show on a black background as grayish-yellow spots, and can either be examined at once under a low



FIG. 26.—Elastic fibers (after Strümpell).

power or the uppermost piece of glass is slid along until the fragment is exposed, when it is picked out and placed upon the ordinary microscopic slide" (Osler).

The form and appearance of the elastic threads differ materially according to their special source. If they come from the alveoli, there is an interlacing of the fibers which may preserve the globular contour of the air-cells. If they come from the blood-vessels, they are single and elongated, or two or three of the fibers may be arranged side by side. Elastic tissue derived from the bronchi presents much the same appearance as when it comes from the vessel-walls.

The presence of elastic fibers furnishes incontestable proof that destruction of lung-tissue has taken place. To show that this loss of structure, however, is due to tuberculosis, we must exclude abscess (an



exceptional event) and gangrene of the lungs—diseases in which it also occurs.

*Hemoptysis.*—This symptom of phthisis will be spoken of under Diseases of the Lungs, but its importance as a diagnostic feature of this disease makes special reference to it here absolutely necessary. It is present in the majority of cases, exhibiting, however, the widest variations both in the amount of blood expectorated and in its frequency of occurrence. The sputum may be merely blood-stained or the hemorrhage may be excessive and prove rapidly fatal, though hemoptysis is rarely the direct cause of death in tuberculosis. Slight hemorrhages are usually produced by mere hyperemia, and are most apt to occur during the early stages, while severe bleedings are produced by the erosion of a blood-vessel or rupture of a small aneurysm, and are most prone to occur during the stage of cavity. In certain cases hemoptysis is frequent.

A third or capillary form of hemorrhage may occur in phthisis with cavity-formation, and in this variety, which is of a rather frequent occurrence, the purulent sputum is uniformly stained with blood. It may also be nummular, but presents a reddish-brown or chocolate color. The exciting cause is seldom obvious, though in not a few instances aggravation of the cough, and in others great mental excitement, would appear to excite bleedings. Slight hemorrhages often, and severe ones rarely, afford more or less relief to the pulmonary condition. On the other hand, severe bleedings usually exert an unfavorable influence, being followed by debility and anemia. Moreover, in numerous cases hemoptysis is followed by a more rapid extension of the local lesions, with corresponding aggravation of the local and general manifestations. The fact remains, however, that the effect of severe hemoptysis upon the progress of chronic phthisis is by no means always untoward. In a case of my own there occurred periodically copious spontaneous bleedings (in spring and fall) for three years, which were as regularly followed by marked improvement for a period of three or four months. The physical signs of phthisis were absent until after that time, when a small area of consolidation was detected near the left apex. The patient was a male aged twenty years, and was sent to the Adirondack region, where the hemorrhage failed to recur and he made a complete recovery.

*Dyspnea* is present, but is not a marked feature, as a rule, despite advanced pulmonary lesions. Perhaps the chief reasons for a lessened demand for oxygen on the part of the system are—first, the slow and gradual manner in which the lesions develop; and second, the pronounced bodily wasting. The respirations, however, are moderately increased in rate, averaging from 20 to 30 per minute, and this compensates admirably for the diminished breathing-space. The dyspnea may be greatly intensified, however, as the result of intermittent pneumonia, pleurisy, active exertion, or great mental excitement, and toward the close of fatal cases the most intense dyspnea may be manifested.

*Physical Signs in the Stage of Consolidation.*—*Inspection* gives most important results. The paralytic or phthisical thorax is generally presented to view. It is flat, particularly the upper half; the intercostal spaces are wide; the ribs slope at a sharp angle from the sternum, mak-

ing the epigastric angle acute and producing elongation of the chest. The same sharp inclination downward from the vertebral column is observed latterly and posteriorly. The angle of Louis is prominent, and the depressions (supra- and infraclavicular, intercostal, etc.) are deepened, the costal cartilages being often prominent and the sternum, particularly in the lower part, sometimes much depressed or even concave (funnel-breast). The scapulæ stand out prominently and may be distinctly winged. A second type of paralytic thorax is narrow and long. Pulmonary tuberculosis may, however, arise in chests of apparently normal build. With the development of phthisis at the apex the depressions of the side affected are relatively deeper, while the clavicle often stands out more prominently. The paralytic thorax is often a resultant of developed phthisis, and occurs in subjects in which the chest was normal, preceding the invasion of the disease. Finally, both narrowing and flattening of the upper parts of the chest may result from great emaciation.

Defective expansion is observed early, and usually at the apex of the side first affected; subsequently this may be more general, and finally bilateral. To note the motions of respiration with precision the examiner should occupy a position exactly in front of the median line of the patient's body. The difference in the movement of the two sides often becomes more apparent on deep respiration than on quiet breathing, and while at rest the respirations are almost normal, but exertion decidedly increases their frequency.

*Palpation.*—Testing the expansion by palpation gives better relative results than does inspection. To determine the relative movements of the apices the extended hands should be so placed (by allowing them to diverge below) that the tips of the fingers touch the lower border of the clavicle, and then the patient should be asked to breathe deeply, though slowly. The expansion in the supraclavicular spaces is tested by standing behind the patient and using the tips of the fingers, or by allowing the two first fingers of each hand to pass parallel with the clavicles. In this way "lagging" over the apex will be the first symptom recognized, and may for some time be the only one.

Tactile fremitus is early increased with oncoming consolidation, due to the growth of the tubercles, though it is normally more marked at the right than at the left apex. If there be great thickening of the pleura, however, it is more or less diminished, and if there be pleural effusion, it is usually absent.

*Mensuration.*—The difference between the measurement of the chest in inspiration and expiration in any person of average health should be not less than three inches, and a difference below two and a half inches points strongly to tuberculosis.

*Percussion.*—Resonance is deadened more and more as consolidation progresses. If the consolidated areas are minute, however, the percussion-note may be unchanged, and as the air-cells surrounding the latter are often emphysematous and relaxed, the note may be somewhat tympanitic. In many cases the tympanitic sound and deadness are intermingled, giving rise to the so-called tympanitic deadened sound. Slight dulness is, as a rule, noted first below the clavicle, though in not a few cases it is first detected upon and above the clavicle. The corre-



sponding regions of the two sides must be compared during a held inspiration, and also during a held expiration. The degree of dulness can sometimes be better estimated by comparing the apical note with that obtained lower down on the same side, allowing for the normal topographic differences of intensity. The latter method is especially applicable to cases in which both apices are involved. Impaired resonance may be detected early in the supraspinous fossa, and less frequently in the interscapular space if the subject be not too stout, though slight dulness in the absence of other signs has little diagnostic value. As the lung-tissue becomes airless throughout an area of considerable size it is markedly deadened, until dulness is heard; finally, with extensive consolidation the note may be wooden and the feeling of resistance be much increased.

*Auscultation.*—The vesicular breathing may be sharpened, owing to narrowing of the smaller bronchi, but more often perhaps it is diminished by the swelling and secretion. The corresponding regions on the two sides must be compared—first during quiet, and then deep breathing, and it should be remembered that prolonged expiration is an early and important diagnostic mark, at first being somewhat sharpened, and later distinctly bronchial. Tuberculous bronchitis may cause interrupted or jerking inspiration at the apex. If heard elsewhere, little value is to be given it. With lobular consolidation at different points in the region affected, the conditions favor the transmission of the bronchial sounds, but these are toned down by the remaining intact air-cells; hence there is “transition” or broncho-vesicular breathing. With complete consolidation, pure bronchial breathing is audible, and with the latter two forms of breathing crepitant or subcrepitant râles are heard. Sometimes the first râles have a low whistling sound, which accompany the long expiration; with liquefaction they become more moist, are louder (sometimes ringing), and often bubbling, and may be heard on inspiration and expiration. If scanty, they may be audible on inspiration only, though they are increased by coughing. If the moist crepitant and subcrepitant râles, often due to concurrent bronchitis, be very numerous, the breath-sounds will be obscured, but after free expectoration as the result of coughing the exact quality of the breath-sound is appreciable.

*Pleuritic friction-sounds* may be heard, due to accompanying pleuritis sicca, and these may be audible before the bronchial râles reveal the disease. Friction-sounds and râles often occur together. *Pleuro-pericardial friction* is present when the “lappet” of lung over the heart is affected, while clicking râles, occasioned by the heart’s systole, are audible when the same area is pneumonic. The vocal resonance increases with the progress of the consolidation, and when the latter is complete *bronchophony* (rarely *pectoriloquy*) is present. In the subclavian arteries a systolic murmur is not uncommonly heard, the latter being supposed to be due to pressure exerted by the thickened pleura upon these vessels.



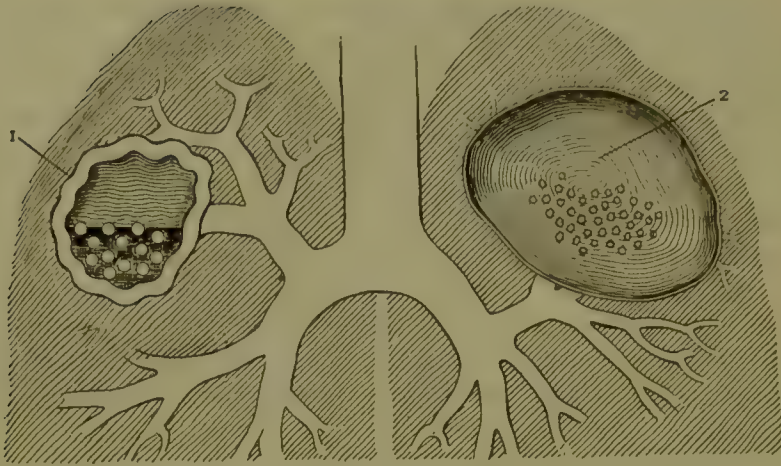


FIG. 27.—1. Small cavity near periphery, with thick relaxed walls, containing secretion and communicating with a bronchus (*vide* subjoined table). 2. Large parietal cavity, with thin, tense, smooth walls, communicating with a bronchus (*vide* table).

#### *Physical Signs.*

- (a) Percussion—deadness on a strong blow, mere impairment of resonance on a light blow; Wintrich's interrupted change of sound, detectable when patient is upright, but not when recumbent.
- (b) On auscultation low-pitched cavernous (hollow) breathing; gurgling (ringing) râles.
- (c) Pectoriloquy indistinct, owing to small size of cavity and the contained fluid.

#### *Physical Signs.*

- (a) Amphoric percussion-resonance, cracked-pot sound, and Wintrich's change of sound.
- (b) On auscultation, high-pitched amphoric (musical) respiration and metallic râles.
- (c) Amphoric (musical) voice and amphoric whisper.

**Physical Signs of Cavity.**—*Inspection* shows a more marked retraction and a more decided lack of local motion than during the previous stage. The degree of shrinking is proportional with the extent of fibrous-tissue formation.

*Palpation* corroborates inspection as to lack of motion, and gives increased tactile fremitus if the cavity connects with an open bronchus and if it contain but little secretion. Excessive secretion interferes with conduction of sound.

*Percussion.*—Resonance is generally more or less impaired in consequence of the consolidation of the surrounding lung-tissue. The note may be somewhat tympanitic, but varies with the position of the cavities, the amount of fluid secretion contained by them, the condition of their walls, and the vibratory capacity both of the latter and of the individual thorax. Cavities of the size of a walnut situated in the apices usually give a distinctly tympanitic note, while cavities of the same dimensions, or even larger, in the lower portion of the lung do not. The metallic tone is especially noticeable over large cavities with smooth walls. The tympanitic sound may be deadened by closure of the connecting bronchus and by temporary filling of the cavities with secretion, and, again, if they are surrounded by thickened lung-tissue or by a large thickened pleura, there may be impaired resonance or absolute dulness even. Certain *special conditions* change the tympan-

itic sound over a cavity. Thus the note will be louder and exalted in pitch when the mouth is opened wide, and lowered when the mouth is closed (Wintrich's sign), there being dulness when the mouth is closed and tympanitic resonance when the mouth is open. If the cavity communicates freely with the bronchus, it may be necessary to change the position of the patient, and a tympanitic note may change in pitch with change in posture (Gerhardt's change of sound). If the patient changes from the dorsal to the upright position, resonance may give way to more or less flatness, since the fluid contents of the cavity are thus brought into contact with the chest-wall, and, although an almost certain sign of a cavity when present, it is exceedingly rare. The so-called cracked-pot sound is often elicited over large parietal cavities with thin walls, and may be quite intense; but, since it also occurs in many other pathologic conditions, its diagnostic significance in this disease is subordinate. There may even be normal resonance if the cavity is covered by a layer of unaffected air-cells of considerable thickness.

*Auscultation* over small vomicæ with lax walls reveals *cavernous* (low-pitched) breathing, while over large cavities with tense walls (if parietal and communicating with a tracheo-bronchial column of air) it gives *amphoric* (higher-pitched) respiration. Moist râles (bubbling and gurgling, according to the consistency of the secretion) may be present, and these correspond in the main to the amphoric breathing, hence being heard most frequently over large, smooth-walled and peripherally-located cavities. The gurgling and slushing sounds caused by the air bubbling through the secretion in a cavity are always intensified by coughing.

*The sounds of falling drops* (metallic tinkling) may be heard over large vomicæ with tense, smooth walls containing thin secretion. *Pectoriloquy* and *amphoric whispers* are the vocal sounds heard over huge cavities, and to the latter should be given the greatest diagnostic significance.

**General Symptoms.**—(a) **Fever.**—Whilst the disease is progressing fever is a constant, significant, and, it may be, the earliest, symptom. If a two-hourly record be kept for a few days, from time to time an accurate conception of the course and type of the fever can be formed. In the first and middle stages the highest temperature occurs about 4 or 5 P. M., the lowest about 4 or 5 A. M. The fever may be continuous, remittent, or intermittent, and in a general way these types, in the order named, correspond to the stages of tuberculization, softening, and cavity-formation. Modified types, due to the fact that the lesions often and simultaneously represent different stages, are also observed. Apyrexial periods are met with in the early as well as the late stages of chronic phthisis, and indicate cessation of the processes of tuberculization and caseation.

*A continued fever* is most apt to be met with during the initial period of phthisis, the evening temperature sometimes registering but a degree higher than the morning. A similar curve may be presented at any later time if acute pneumonia supervene, though it is to be recollected that the remissions in such cases are usually greater than in primary lobar pneumonia.

*A remittent fever* is more common than the preceding type. It may be present from the start, but is oftener seen in the middle, and less fre-

quently in the advanced, stages of phthisis. This form of fever points to softening (see Fig. 28).

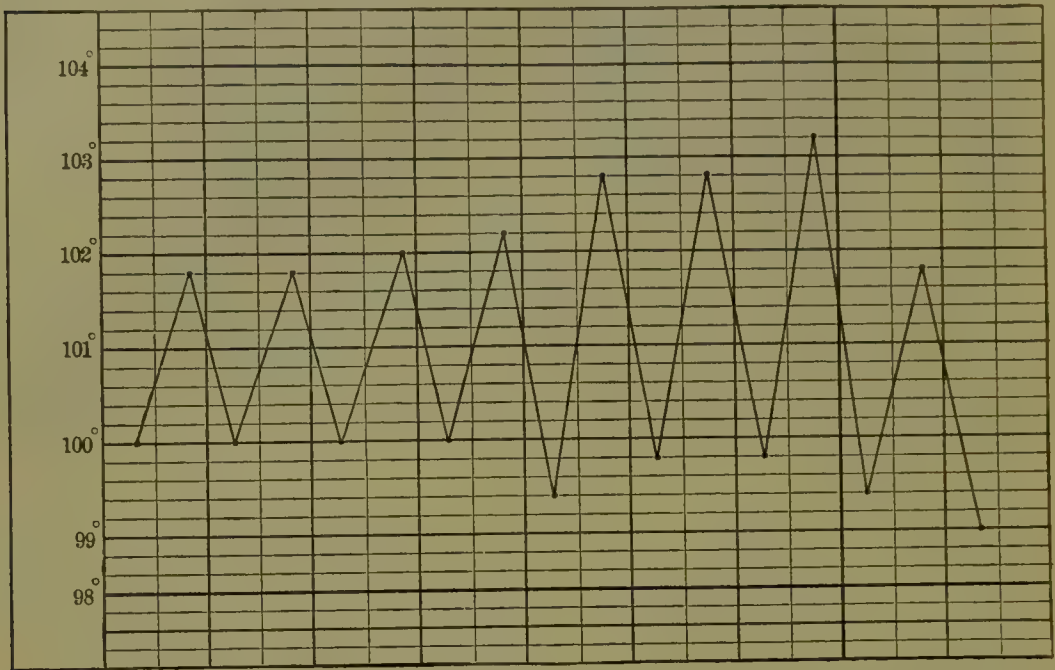


FIG. 28.—Temperature-chart of a case of phthisis. Quiescent cavity in right apex, and commencing excavation in left apex. Robert G—, aged 21 years; dyer.

An *intermittent fever* is also frequent, and is characteristic of cavity formation, suppuration being invariably associated with the latter process.

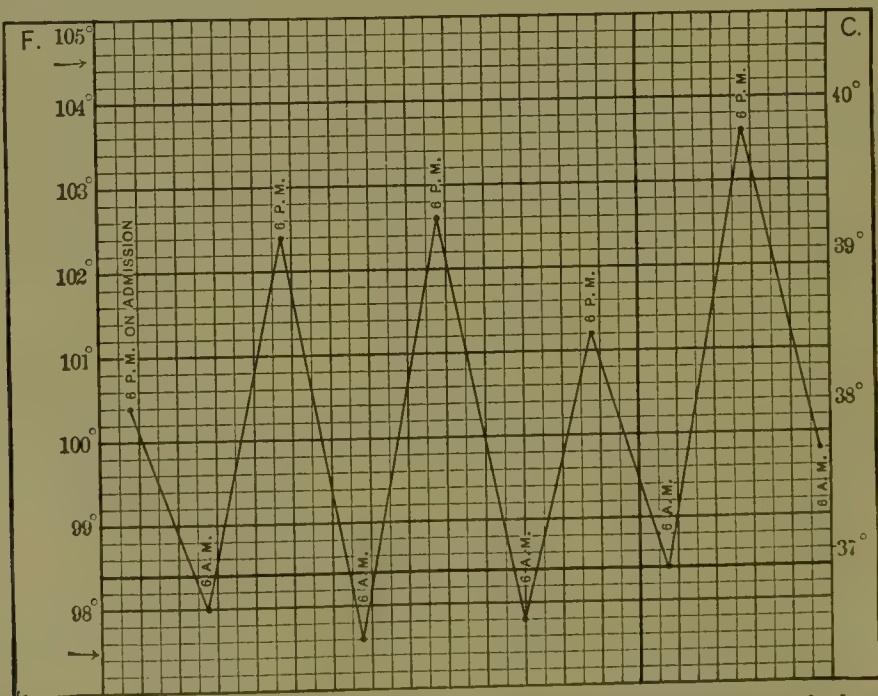


FIG. 29.—Temperature-chart of a case of phthisis. Cavity in left apex, giving cracked-pot sound, Wintrich's sign, etc. George C—, aged 22 years; glass-worker.

The temperature may be intermittent from the start, suggesting malaria to the unguarded; but it is due to sepsis, the temperature rising during



the day, beginning usually shortly before noon, and reaching its maximum at from 5 to 8 P. M. It now falls slowly until about 4 or 5 A. M., and then rapidly reaches the minimum—a subnormal point—usually at from 6 to 10 A. M. For a considerable portion of every twenty-four hours the temperature may be below the normal, sometimes dropping as low as 95° F. (35° C.). (See Fig. 29.)

(b) **Night-sweats** occur in a large majority of the cases. They may appear during any part of the course of phthisis, though most apt to occur and be most marked during the progress of cavity-formation; they show themselves in the early morning hours simultaneously with the rapid decline in the temperature, and may appear during sleep at any period of the day. They may be light and limited to the neck and upper portion of the thorax; on the other hand, they are often excessive, saturating the bed-clothes and inducing great exhaustion. The drenching sweats are dependent partly upon the fever and partly upon the existing weakness, though slight exertion may also engender free perspiration.

(c) **Emaciation** occupies a prominent place in the symptomatology, the muscular and fatty tissues being involved to an equal degree (Strümpell); the extremities and soft parts of the thorax are most affected. It must be remembered that an exalted grade of emaciation may be present at an early period, and in such cases it may be assumed that the thinness of flesh was a precursory state. In nearly all cases an extreme degree of emaciation, reducing the patient to a slightly covered skeleton, is reached before the end. The causes of emaciation are chiefly the persistent fever, the loss of appetite, and the feeble digestive and assimilative powers. It is an almost invariable rule that during the afebrile periods, associated as they are with improved appetite and digestion, the patient gains in flesh and strength.

(d) **The pulse** is rarely increased in frequency, is of good volume and regular in rhythm, though of low tension (soft). When suppurative fever sets in the pulse becomes very frequent, small, and compressible, and the capillary pulse is often observed; rarely venous pulsation may be noted in the back of the hands.

(e) **Anemia** is one of the symptoms evidencing impaired nutrition, and seems to appear in certain cases before the more obvious local lesions. The objective changes pointing to anemia are pronounced (pallor of lips, visible mucous membranes, and skin). The *blood* presents nothing characteristic. In the early stage it may be normal, or in some cases chlorotic in type, the hemoglobin being decidedly deficient; later in the disease, when consolidation is advanced, the blood may be wholly normal; but when there is cavity-formation and hectic fever, there is often a very considerable leukocytosis, as many as 50,000 leukocytes per cubic millimeter having been observed. The differential count shows a great excess of the polynuclear cells. It is not unlikely that the condition is due to secondary infection by the pyogenic micro-organisms, and especially by the streptococcus.

*General debility* is complained of in all cases. It is usually progressive and may amount to a feeling of utter exhaustion.

**Symptoms and Complications presented by Other Organs.**—(a) **The Heart.**—The pulse and the blood-appearances have already been described. With retraction of the upper lobe of the left lung the area of the heart's impulse

is obviously increased, particularly upward, so that pulsation may be visible in the fourth, third, and even second interspaces, near the sternum, while the normal apex beat in the fifth interspace may be wanting. Systolic murmurs both at the apex and the pulmonary orifices are often audible, and occur independently of valvular lesions, though the latter may supervene or may constitute an associated condition. Chronic endocarditis affecting the tricuspid segments is not infrequent in phthisis.

(b) **Gastro-intestinal Tract.**—The tongue may be furred: more often it and the mouth and throat are red, showing increased irritability. The pharynx may be the seat of tuberculous lesions, which may interfere greatly with deglutition, *aphthous ulcers* being common, while *thrush* may also appear in the later stages. The appetite is impaired or lost: thirst is annoying, and the symptoms of subacute and chronic gastritis (interstitial and parenchymatous) often obtain. With the latter is not infrequently associated a catarrhal ulceration, and with equal frequency, perhaps, dilatation; and the stomach may be so irritable that the presence of food, or even coughing, at once excites vomiting: this symptom is most troublesome during the third or last stage of the affection. A study of the gastric secretion gives variable results, there being an early hyperacidity, while later the secretion is subacid.

The causes of gastric symptoms are not clear. The mucosa is the seat of venous engorgement, and may thus occasion the catarrhal changes that are present in many instances. There are not a few cases, however, in which the symptoms are serious without adequate local anatomic changes to explain them.

*The intestinal symptoms* are but little less important than the gastric. During the early stage constipation is a frequent condition, and yet few cases run their entire course without manifesting diarrhea. The latter symptom may occur at any time, but is most prone to appear at an advanced period, and may pursue an intermittent course. Occasionally it alternates with periods of "hectic fever," and late in the affection a watery discharge may develop (*colliquative diarrhea*). The intestinal lesions are of three sorts: (a) *catarrhal*, (b) *ulcerative*, and (c) *amyloid*. These often arise in the order enumerated, but are not infrequently combined in various ways. Hemorrhoids and anal fistulæ are among the common complications.

(c) **Genito-urinary Organs.**—There is frequently an albuminuria that may either be of an ordinary febrile nature or due to chronic nephritis (*productive and non-productive*). *Chronic nephritis* is usually a late development, and is associated with a rather marked albuminuria, the presence of tube-casts in the urine, and dropsy. *Amyloid changes* may also set in toward the close, attended by their characteristic urinary phenomena. As secondary events tuberculous *pyelitis* and *cystitis*, with the appearance of pus and rarely blood in the urine, may develop. Hematuria may also result from temporary congestion. The testes may be implicated, and a routine inspection of these organs, as suggested by Osler, should not be neglected.

(d) **Cutaneous System.**—Cyanosis occurs, but, being of a moderate degree, it is often veiled by a decided pallor. The cheeks often wear a "hectic flush," and the skin, late in the affection, is apt to be dry, harsh, and scaly. Among the cutaneous appearances are pigmentary stains over



the chest (*chloasmata phthisicora*), and seated in the same regions as well as upon the back are frequently seen brown stains (*pityriasis versicolor*). The hair over the chest often becomes gray; that of the head and beard, long and harsh. The finger-ends are often bulbous (clubbed), with incurved nails, though this is not peculiar to chronic phthisis, and cracking of the finger-nails is also often observed.

(*e*) **Nervous System.**—The mental attitude is characteristically hopeful and buoyant, even in the advanced stages. Hence the patients are readily encouraged by the unscrupulous to believe that their condition is improving, despite the steadily unfavorable progress of the disease, and, indeed, they may be in an utterly helpless state, and yet confidently expect to recover. The cerebral symptoms are rarely marked, and the mind, as a rule, is exceptionally clear. Tuberculous meningitis and meningo-encephalitis may develop secondarily, and I have met with cerebrospinal meningitis in one of my own cases. Osler has seen two cases in strong, robust men “in whom the existence of pulmonary phthisis was not discovered until the post-mortem.” Focal lesions, due to the presence of tubercles, may produce forms of paralysis (aphasia, hemiplegia, etc.) according to their location. Rarely peripheral neuritis (usually an extensor paralysis of the leg) is observed. Insanity, quite independently of nervous lesions, is a rare complication.

(*f*) **Chest-muscles and Mammary Glands.**—The former are abnormally irritable, and sometimes even painful on percussion, and the mammary gland is in rare instances hypertrophied, males suffering most; but, as pointed out by Allot, the affection is a chronic non-tuberculous mammitis, and hence a true complication.

**Diagnosis.**—The early recognition of chronic pulmonary tuberculosis often tests severely the diagnostic acumen of the physician. The general and local symptoms, including the physical signs, may afford merely a strong suspicion of the existence of phthisis, and in such instances repeated examinations of the sputum for the bacilli are imperative, and only when they are found is the diagnosis set at rest. Repeated staining of the sputum may be necessary for the detection of tubercle bacilli. It is also desirable to determine whether they are constantly present by re-examinations at intervals. There are not a few cases in which the physical signs are clearly obvious, and in which the bacilli are either not at all detectable or only after several examinations; and the fact that a certain diagnosis is made possible only by the demonstration of the bacilli in the sputum in the incipient stage of the affection makes a continued search for these micro-organisms the first duty of the physician in cases presenting suspicious signs and symptoms. An absence of the bacilli, however, does not justify a denial of the existence of phthisis, and is of little negative value. In a certain percentage of cases the careful study of the symptoms and general course of the disease is of paramount importance; and of these symptoms the most valuable are cough, expectoration, fever, progressive emaciation, and the constant presence of certain physical signs situated at the apex on one side (flattening of the front of the chest, defective expansion, slight deadening of the percussion-note, and a change in the vesicular murmur, with or without adventitious sounds). The diagnostic import of elastic fibers in the sputum is also to be borne in mind. The physician should appreciate



fully his own grave responsibility in all cases of suspected phthisis, and earnestly endeavor to determine the diagnosis early while the condition yet admits of cure.

In the more advanced stages of phthisis the diagnosis is rarely dubious or even difficult, and is made usually from the characteristic features—local and general—which are confirmed by a microscopic examination of the sputum.

**Differential Diagnosis.**—In the very early stage the local condition may be obscured by the general features, and most frequently by the fever, though it may also be by the evidences of anemia or the gastric symptoms, with a falling off in the general health. The danger of confounding these conditions with phthisis is slight if the various modes of onset previously described be kept in remembrance.

*Bronchial catarrh* is with great difficulty discriminated from beginning phthisis. If the temperature is elevated from 2 to 5 P. M., and not at all or only slightly above the normal night temperature in the evening, the probabilities are greatly in favor of tuberculosis (Barlow). The condition of the lungs must be carefully compared, since in bronchial catarrh there is no dulness, and moist râles, that vary in intensity from one day to another, are heard equally on both sides. From time to time râles may also be heard at the bases in bronchitis. In phthisis one apex is more involved than the other, the moist sounds not being heard equally low, and after repeated coughs with subsequent deep inspiration the râles are more apt to remain than in ordinary bronchitis. In phthisis, also, there is a gradual loss of flesh and strength—a feature that is absent in bronchitis. In diagnosing between these affections in doubtful cases it is of the utmost importance to repeatedly examine the sputum for tubercle bacilli.

Phthisis in the stage of cavity may be confounded with *bronchiectasis*. The points of discrimination are, however, given under the latter affection (Diseases of the Lungs).

### FIBROID PHTHISIS.

**Definition.**—Fibroid phthisis implies induration followed by contraction of the affected lung-tissue, due to an increase in the connective-tissue elements. There are cases in which it cannot be distinguished pathologically from chronic pulmonary phthisis, but from a clinical point of view the two affections present distinctive peculiarities. The majority of instances are primarily tuberculous, though manifesting a strong tendency to the formation of fibrous tissue—a conservative process; in other instances the fibroid change may be primary, followed by tuberculous infection (*vide* Pneumonokoniosis). The usual form arises variously as a sequel of other morbid processes, such as—

- (1) Pneumonias, acute lobar (very rarely) and catarrhal pneumonia (commonly).

- (2) Pulmonary lesions, such as a tubercle in the stage of consolidation or cavity.

- (3) Chronic tuberculous pleurisy.

- (4) Bronchial catarrh from inhalation of irritants (steel-, coal-, or mineral-dust). As stated under the description of pneumonokoniosis

(*vide*), this condition may be found to be a tuberculous process accompanied by fibroid change.

**Pathology.**—The process in the beginning is very often localized in one apex, and less frequently in the middle portion of the lung or in the bases. It may remain circumscribed, but more often it extends downward, and gradually invades the entire lung. It is unilateral. Secondary to the induration and contraction there is dilatation of the bronchi, and rarely bronchiectasis may precede the fibroid induration.

The lung-tissue is hard and dense, the alveoli being obliterated. It resists cutting and creaks on pressure, and the section presents a smooth, dry, gray, often marbled aspect, though the fibrous tissue may undergo caseation. Tuberculous lesions may also develop in the opposite lung and in still other organs.

The pleura is thickened as a rule, often to a marked degree, and its layers are adherent; the unaffected portions of the lungs frequently become emphysematous. The right ventricle is, as a rule, hypertrophied.

**Symptoms.**—These may be briefly stated, since they do not differ from those of cirrhosis of the lung, which is described in the section on Diseases of the Lungs. The onset is extremely insidious: a persistent cough that is apt to occur in severe paroxysms in the mornings, and a purulent expectoration, are for a long period the leading features. If bronchiectasis is present, the sputum may be fetid. Dyspnea is marked, particularly on exertion. Fever is slight or absent, and hence emaciation progresses slowly or may not even be noticeable.

The *physical signs* are obvious, and are identical with those of fibroid induration of the lung (*vide infra*).

The course of this disease is exceedingly long, ranging from ten to twenty or even thirty years, and, as I have before stated, both lungs may become the seat of tuberculous disease. Again, as in chronic pulmonary tuberculosis, prolonged suppuration may lead to amyloid changes in the liver, spleen, kidneys, and intestines. Dropsy, due to secondary dilatation of the right ventricle, often closes the scene.

**Differential Diagnosis.**—*Chronic bronchitis* is prone to be mistaken for fibroid phthisis. In the latter disease, however, there are unilateral retraction and the signs of consolidation of the apical cavity, all of which symptoms are absent in chronic bronchitis.

**Complications.**—*Lobar pneumonia*, and less frequently *lobular pneumonia*, may develop and cause a fatal termination, and doubtless the fact that it is impossible to discriminate these conditions from an acute tuberculous pneumonia has frequently led to confusion as to the exact nature of the cases.

*Erysipelas* may arise in the course of chronic pulmonary tuberculosis, though the proportion of cases is not formidable. Out of 1165 cases of erysipelas, 15 coexisted with pulmonary phthisis.<sup>1</sup> Some contend that its occurrence in this disease may be beneficial, but my own observations tend to show that the gravity of both conditions is increased when occurring together.

*Typhoid fever* may rarely be met with in sufferers from chronic phthisis, though I have seen but a single instance come to autopsy.

<sup>1</sup> "Points in the Etiology and Clinical History of Erysipelas," *Journal of the American Medical Association*, July 2, 1893.

In another fatal instance the characteristic symptoms of typhoid were present during life, but no post-mortem examination was allowed. It is important, however, not to overlook this occasional association of two such common affections.

*Chronic nephritis* and pulmonary tuberculosis are often found in the same subject, and with these arterio-sclerosis is quite commonly combined.

*Chronic endocarditis*, particularly of the tricuspid segments, may also occur in phthisis, and from time to time cases of valvular heart-disease are reported, in which it is evident that passive congestion must have existed for some time before the tuberculous condition developed. The old doctrine of the mutual antagonism between disease of the left heart and pulmonary tuberculosis finds not a little support from these cases, as in a large proportion a very considerable tendency to encapsulation of the lesions exists.

**Course and Duration.**—Both as to course and duration this disease exhibits unusual variations. If not promptly treated during the time in which there is hope of therapeutic and climatic efficiency, it frequently progresses with more or less rapidity toward the grave. On the other hand, it is common to observe periods during which the disease is arrested or improved. Generally, the improvement, though followed by an exacerbation, endures for a long time, and permanent cures, even in the advanced stage, are by no means rare. The duration of pulmonary tuberculosis varies exceedingly, though from the collective investigations of different authors and from all the statistics available I find the average duration to be about three years. The late Austin Flint long ago directed attention to the innate tendency of a considerable percentage of the cases to spontaneous recovery—a fact that simply indicates a victory for nature's silent defensive processes in the struggle for existence.

In fatal cases death is by (a) *gradual asthenia* (most frequently), with retention of consciousness until the end approaches.

(b) *Complicating conditions* (bronchitis; pneumonia; pleurisy; pneumothorax; amyloid degeneration of the intestines, liver, spleen, kidney; Bright's disease; diabetes, etc.).

(c) *Tuberculosis of other organs*, particularly the meninges, intestines, and genito-urinary tract.

(d) *Hemorrhage*, due commonly to rupture of an aneurysm in the lung-cavity; less frequently to erosion of a large vessel. Fatal hemorrhage may, when the vomica is of large size, occur without hemoptysis, as in a case of Roland G. Curtin's at the Philadelphia Hospital.

(e) *Syncope*.—Though of comparatively rare occurrence, there are a number of events that may lead to sudden, fatal syncope—*e. g.* hemorrhagic embolism or thrombosis of the artery, pneumo-thorax, thoracentesis for pleural effusion, walking about in a moribund state, etc.

(f) *Asphyxia* rarely closes the scene in acute pneumonic phthisis, and very rarely in chronic phthisis complicated with pneumo-thorax, or with a large undiscovered or neglected empyema, or with sero-fibrinous pleurisy.

#### TUBERCULOSIS OF THE ALIMENTARY TRACT.

(1) *Lips*.—Whilst tuberculosis of the lip is quite rare, the possibility of its occurrence must not be forgotten. In a case of my own it assumed,



as is usual, the form of a small ulcer, and the diagnosis was made by an examination of the labial mucus. It followed an accidental lesion of the lip from biting, and was not associated, as are most cases, with laryngeal or pulmonary tuberculosis. In *diagnosing* the condition chancre and epithelioma must be excluded, the former by the history, and the latter chiefly by microscopic examination for tubercle bacilli.

(2) *Tongue, Palate, and Tonsil*.—The work of Orth, Hanan, Schlenker, Kruckman, and others has shown that the tonsils, owing to their frequent inflammation and the destruction of their mucous membrane, are frequently the point of entrance of the tubercle bacilli, and thus of the infection of neighboring glands. The fact that tuberculosis of the tonsils has repeatedly been found, and often when unsuspected from the gross appearance, and when other lesions of the disease did not exist, points to the not infrequent occurrence of primary tuberculosis in this site. The infiltrated areas often present small grayish spots, but the appearance of the ulcers is not characteristic, frequently bearing a strong resemblance to epithelioma and to the syphilitic ulcer. The *diagnosis* demands either inoculative experiments or a microscopic examination of the oral mucus, the latter being oft repeated if necessary.

(3) *Pharynx and Esophagus*.—Both miliary tubercles and ulcerative lesions may rarely arise on the posterior wall of the pharynx by direct extension from laryngo-pulmonary tuberculosis or as the result of secondary inoculation. The chief symptoms occasioned are the excessive secretion of pharyngeal mucus and muco-pus, and painful deglutition. Tuberculosis of the esophagus is extremely rare. Osler, however, saw a case in his wards in which the ulcer perforated the esophagus and caused purulent pleurisy.

(4) *The Stomach*.—Tuberculous lesions appear only exceptionally in the mucosa of the stomach, notwithstanding the fact that marked gastric symptoms are of frequent occurrence. It should not be forgotten that nausea, vomiting, and other gastric symptoms may be directly due to involvement of the larynx. I have been able to find reports of 4 such cases in addition to the 12 collected by Marfan.<sup>1</sup> The ulcers may be single (as in Musser's case) or multiple (as in Osler's case). The symptoms are not characteristic, but hematemesis occurring in patients suffering from tuberculosis of other organs should excite a strong suspicion of the existence of ulcer. Pain coming on soon after meal-time is more marked in tuberculous ulcer of the stomach than in ordinary gastric lesions. Perforation has taken place in some cases, with its usual dire consequence.

(5) *Intestines*.—The lesions may be (a) primary, or (b) secondary to tubercles of other organs (lungs, peritoneum, etc.).

(a) Primary tubercle of the intestines is chiefly met with in children, for the reason that they are more apt to swallow the tubercle bacilli with their food, and especially in milk. But though this form of the disorder occurs much less frequently in adults than in children, the intestinal route of infection is, according to my own observation, more common than is generally supposed. Many cases during life present the features of both intestinal and peritoneal tuberculosis, and it is often impossible to determine which of these was the primary condition; and the same difficulty arises when the cases come to autopsy. Yet I have never seen an instance

<sup>1</sup> *Paris Thesis*, 1887.

(post-mortem) in which the peritoneum and mesenteric glands were not involved to an equal degree.

(b) The secondary variety occurs in more than one-half of the cases of pulmonary tuberculosis, the chief seats of the lesions being the lower part of the ileum, the cecum, and the upper part of the colon. The rectum is also the seat of secondary tuberculosis in a small proportion of the cases of chronic phthisis, and it may, with great rarity, be a primary seat of the affection.

The morbid process begins in the solitary glands in Peyer's patches, where at first grayish, firm tubercles grow and form little prominences. These caseate, becoming yellow in appearance, and then soften and disintegrate, producing ulcers. Osler thus describes the characteristics of the tuberculous ulcer: "(a) It is irregular, rarely ovoid or in the long axis, more frequently girdling the bowel; (b) the edges and base are infiltrated, often caseous; (c) the submucosa and muscularis are usually involved; and (d) on the serosa may be seen colonies of young tubercles or a well-marked tuberculous lymphangitis."

The cicatrices are extensive and often pigmented, and as they undergo contraction may produce incomplete or even complete stricture of the bowel. At a point corresponding to the seat of the ulcers, local peritonitis invariably develops. The serosa is thickened and adherent, and the ulcer may penetrate through this coat without causing perforative peritonitis, while rarely a fistulous connection is established between different parts of the intestine. The base of the ulcer may show signs of hemorrhage.

**Symptoms.**—In children the symptoms are those of a protracted catarrh of the intestines. Diarrhea may be a prominent feature, though it indicates involvement of the large intestine, which does not occur as a rule until the lesions in the small bowel have reached a rather advanced stage. More often there is constipation, which may be due to peritonitis, and often there is irregular fever, wasting, and a lack of development. These general symptoms of tuberculosis may antedate the local, but they are especially valuable for diagnosis.

In adults intestinal tuberculosis generally gives rise to symptoms similar to the above, and when they arise in the course of pulmonary phthisis they are highly significant. If diarrhea be present, it stubbornly resists treatment, and it must not be forgotten that this symptom may also be due either to catarrhal colitis or to amyloid change, both of which processes may be associated with chronic phthisis. Constipation is common and often marked, and local tenderness and colicky pains are complained of frequently. The pulmonary signs, however, may be in abeyance.

If the abdominal and general symptoms are such as to excite suspicion of this disease, then a rigid physical examination of the lungs should be made. The chief seat of the lesions may be for a long time in the cecum, or in the appendix, when the symptoms—both local and general—will be those of appendicitis.

The **diagnosis** of primary intestinal tuberculosis is beset with special difficulties. Sawyer<sup>1</sup> has in special instances demonstrated the presence of clusters of tubercle bacilli in the rectal mucus, and in this way the recognition of intestinal tuberculosis at an early date, or before diarrhea

<sup>1</sup> *Medical News*, May 23, 1896.



sets in is rendered possible. The mucus is obtained after placing the patient in a position as if to examine for piles, and directing him to bear down as though at stool, by gently removing a small quantity from the everted membrane with a sterile loop. It is then spread upon a clean cover-glass and treated exactly as sputum in the ordinary examination. The same method is applicable to cases of secondary intestinal tuberculosis, but here the history and associated tuberculous lesions usually serve to remove all doubt.

### TUBERCULOSIS OF THE SEROUS MEMBRANES.

General tuberculosis of the serous membranes *secondary* to pulmonary and intestinal tuberculosis is of common occurrence, and that a *primary* form of tuberculosis of the serous membranes also occurs is undoubted. Unfortunately, accurate means of discriminating the secondary from the primary form are wanting, since often in the secondary variety the primary lesions in other organs are insignificant.

The anatomic alterations resemble those of ordinary inflammation of these structures, plus the presence of nodular tubercles. The latter may be observed, as a rule, only over small, scattered, circumscribed areas, though not infrequently they are both numerous and diffuse (general miliary deposit). The effusion is in most instances sero-fibrinous, though sometimes it becomes purulent subsequently, and not infrequently is hemorrhagic. Most instances of so-called hemorrhagic pleurisy are due to pleural tuberculosis.

Clinically, cases are divisible into (1) acute serous membranous tuberculosis and (2) chronic tuberculosis. The *acute* form results from inoculation of the peritoneum or pleura, induced by limited foci in the bronchial, tracheal, or mediastinal lymph-glands, or in the Fallopian tubes in women. The *chronic* type is apt to result from a direct extension of a tuberculous process from some organ adjacent to the pleura or peritoneum, though it may attack the serous membranes primarily. Belonging to this class of diseases are two groups of cases: those attended by sero-fibrinous or sero-purulent effusion and the presence of caseous masses, and those in which there is a tuberculous deposit with increased density and great thickening of the pleural layers, and slight exudation. The pericardium may be similarly involved.

(a) *Tuberculous meningitis* has been described fully in the present section (*vide* Miliary Tuberculosis).

(b) *Tuberculous Pleuritis*.—This subject will be referred to in the section on Diseases of the Pleura. Its import, however, is such that brief special consideration is demanded, and from a clinical view-point the cases may be grouped under two heads—namely, *acute* and *chronic tuberculous pleurisy*.

The *acute* form often has a sudden onset, the initial symptoms being a rigor or repeated fits of chilliness, a stitch-like pain in the side affected, shallow, catching breathing, a cough, and fever. The ushering-in symptoms sometimes suggest lobar pneumonia, and a fatal termination is not uncommon, though apparent recovery or a transition into chronic tuberculous pleuritis also occurs.

Chronic tubercular pleurisy is vastly more common than the acute



form, and it is sometimes primary, though more often secondary to pulmonary tuberculosis. In all cases of the latter disease in which the periphery of the lung becomes involved the visceral layer of the pleura is invaded. This leads to plastic pleurisy with adhesion, and the membranes contain disseminated tubercles or sero-fibrinous tuberculous pleurisy; but, as above stated, the effusion may be hemorrhagic and may also become purulent. When the tuberculous pulmonary focus perforates the pleural sac, pyopneumothorax is produced. In tuberculous pleurisy, as opposed to simple pleurisy, there is usually an absence of leukocytosis.

**Symptoms.**—The onset is very insidious and often unnoticed. There may be few symptoms, and yet a physical examination reveals a large sero-fibrinous effusion. The cough and other symptoms are frequently due to a coexisting tuberculosis of the lungs, but the presence of subcrepitant and dry râles are strongly confirmatory of tuberculous pleurisy. By and by the evidences of pulmonary tuberculosis are of importance, or the supervention of acute general miliary tuberculosis makes clear the nature of the case. The subacute variety with effusion may terminate, after absorption of the exudate, in chronic adhesive pleurisy with great thickening of the membrane. The latter may also originate as a primary proliferative process.

#### TUBERCULOSIS OF THE PERICARDIUM.

The morbid lesions are analogous to those of tuberculosis of the pleura. The effusion may be enormous on the one hand or insignificant on the other, and it is often hemorrhagic, while in the chronic form there is marked thickening of the membrane with the deposit of tubercles and cheesy masses. The affection is less common than tuberculosis of the pleura, yet not so rare as was formerly supposed, and occurs in the acute and chronic forms.

**Acute tuberculous pericarditis** is rarely a primary affection, and, as a rule, originates secondarily to pulmonary, pleural, or glandular tuberculosis. It is especially prone to arise in tuberculosis of the bronchial and mediastinal lymph-glands, and, as the latter condition is frequent in young children, so tuberculosis of the pericardium is relatively frequent at this period, though it may occur at any time of life. Pericardial tuberculosis also results from direct extension from a contiguous focus. The symptoms will be detailed in the discussion of Pericarditis. In the *diagnosis* of the affection the history and any associated tuberculous processes detectable must be taken into account, and a point of some diagnostic value rests in the fact that tuberculous pericarditis does not show the usual inflammatory leukocytosis.

**Chronic Tuberculous Pericarditis.**—This may be a part of the general tuberculosis of the serous membranes, or it may follow the infection of the bronchial and mediastinal glands (most frequently), lungs, pleura, or peritoneum. Undoubtedly, cases of primary origin also occur, but they are exceedingly rare, the neighboring lymph-glands being very generally involved. This form is also dependent upon direct extension from the lungs, the spine, and sternum.

From personal observation I am convinced that the cases naturally fall under two heads, when considered clinically: those without effusion,

in which the pericardium is adherent; and those with more or less effusion. The former are the more frequent, though often entirely latent, the adherent pericardium leading to hypertrophy of the heart, followed sooner or later by dilatation. The signs are therefore those of adherent pericardium, with the occasional difference that the dulness may extend higher up over the sternum, in consequence of the presence of firm, cheesy masses at the base of the heart and also encircling the aorta. The smaller group of cases (in which the effusion is present) resembles dilatation of the heart in its clinical manifestations. I recall one instance of this sort that occurred in a male aged about sixty years at the Episcopal Hospital, the autopsy revealing extensive pulmonary tuberculosis and chronic tuberculous pericarditis, with the presence of eight ounces of hemorrhagic effusion.

#### TUBERCULOSIS OF THE PERITONEUM.

This is dependent upon infection by means of the bacilli circulating with the blood, or upon extension of tuberculous inflammation or ulceration from adjacent organs. Mention has already been made of the fact that the intestines are often invaded by tuberculosis, and that the serosa is quickly involved in such instances. The condition may rarely be primary. This involvement may remain circumscribed and undergo spontaneous cure if the intestinal lesion cicatrizes, as post-mortem findings frequently indicate, but in extensive peritoneal involvement spontaneous resolution is out of the question. These cases may be subdivided into acute and chronic. The *very acute cases* are those forming a part of acute general miliary tuberculosis, or due to perforation into the peritoneal sac from adjacent organs, and Adlebert's classification is as follows: (a) the ascitic form, (b) the ulcerous form, and (c) the fibroid form. Though these groups do not present sharp clinical distinctions, the courses they run vary considerably, as do the results of treatment. In the *ascitic* form the exudate is purulent or sero-purulent, and is often encapsulated. In the *ulcerous* the tuberculous new-formations, which may be quite large, undergo caseation and ulceration, the latter process being progressive, so that it may perforate the walls of the intestines. This and the *ascitic* form may be combined.

In the third or *fibroid* form the peritoneal surfaces are adherent. There is little if any exudation; the tubercles may be numerous and diffuse, or may be found only in scattered localized areas, and are often pigmented. The lesions may represent the concluding stage of acute or subacute tuberculous peritonitis.

**Etiology.**—Most cases are produced by extension of tuberculous inflammation from adjacent organs, and of 107 cases analyzed by Phillips the lungs were involved in 99, the pleura also in 60, and the bowel in 80. Children are frequent victims to intestinal tuberculosis, and the bacilli often reach the peritoneum through the intestines, as they are also apt to do in adults suffering from chronic phthisis. Extension from the pleura to the peritoneum is frequent (pleuro-peritoneal), but from the pericardium it is relatively infrequent. In females the starting-point is very often the Fallopian tubes, and in either sex it may be the appendix.

**Predisposing Factors.**—*Age.*—During the period from fifteen to forty



years the incidence is most frequent, though it is not uncommon in children under ten years, nor between the fortieth and fiftieth years of life. Subsequently, it rapidly decreases in frequency. I agree with Osler in stating that in America negroes are more prone than whites.

*Sex* has a tolerably potent disposing influence. Abdominal surgeons have taught us that the disease occurs more frequently in females than males, owing to the fact that the Fallopian tubes are a favorite seat for primary tuberculous infection. The ratio based upon sex is as 3 to 2 in favor of females.

**Symptoms.**—Some cases develop abruptly with severe symptoms, as fever, marked constitutional disturbance, rapid small pulse, abdominal pain, vomiting, and sometimes diarrhea. The temperature may be quite high ( $103^{\circ}$  to  $104^{\circ}$  F.— $40^{\circ}$  C.), or it may be only slightly elevated even in the worst cases. There follow quickly such symptoms as anemia, marked emaciation, and a pronounced typhoid condition. The signs of peritoneal effusion (rarely large) are soon in evidence, and are attended sometimes by a suppurative type of temperature, sweats, etc., indicating the presence of pus in the peritoneal sac. A few cases are unattended by ascites, and here nodular masses are palpable, while on auscultation friction-sounds may be audible in the umbilical region. Tympanites, due to intestinal paresis, is common in cases having an acute onset.

The *acute stage* may be absent, the affection then being marked by slight local and general symptoms (low fever, anemia, slight belly-pains, and a sense of distention). The skin is sometimes pigmented, and usually in patches. There are not a few instances in which the affection is latent, and in one case of this sort with ill-defined general symptoms pigmentation of the skin first directed my attention to the peritoneal condition.

The **physical signs** of moderate ascites frequently, and those of enlarged mesenteric glands sometimes, are present. These conditions are often combined in children, constituting the so-called *tabes mesenterica*. I cannot conceive of the occurrence of this association of symptoms without simultaneous involvement of the peritoneum, and doubtless tuberculosis of the latter membrane and intestines usually coexists. The tuberculous new growth in the peritoneum may also form a distinct tumor not unlike that produced by glandular enlargement, while the intestinal coils with their now thickened walls are sometimes knotted together so firmly as to simulate a dense new growth. The exudation may be loculated owing to adhesions between peritoneal layers of the intestinal coils, etc., producing a localized tumor varying in size and position. Such sacculated exudations most frequently occupy the pelvic or umbilical regions, though they may also be found elsewhere in the abdomen. They may be multiple, and are not infrequently too small to be recognized by the physical signs, being often discovered during laparotomy. On the other hand, they may occupy a large portion of the abdomen. An *omental* tumor of characteristic elongated form (produced by a shrinking and curling up of this membrane) is demonstrable, its long axis generally taking a transverse direction just above the umbilicus. Gairdner has observed this tumor to disappear by spontaneous resolution in children.

The *dry, fibrous variety*, which is not infrequent, is often latent, and the condition may be general or localized. It is decidedly more frequent



in adults than in children, though the *symptoms* are far from characteristic. Among local features are pains, abdominal distention (giving rise to a tympanitic note on percussion), tenderness on pressure, and sometimes a tumor-ridge extending across the upper abdominal region. Among general symptoms are usually anemia and emaciation, with or without fever. Indeed, the temperature may be subnormal, and these cases may show a tendency to spontaneous recovery.

**Diagnosis.**—Unless tuberculosis of other organs can be demonstrated the diagnosis is often impossible. This is particularly true in cases in which there is no abdominal pain nor tenderness. Fever and the presence of a tumor, especially if the latter be elongated and lies transversely in the umbilical region, are important aids; but if tuberculosis of the lungs, pleura, pericardium, appendix, and the tubes, in women, can be excluded, the rectal mucus and the urine should be examined for tubercle bacilli. From the *acute form* several affections must be discriminated:

(a) *Internal Hernia*.—This comes on suddenly; the pain is strictly localized and paroxysmal; stercoraceous vomiting appears in a few hours; the constipation is absolute, and tympanites is marked, but ascites is absent.

(b) Similar symptoms belong to *volvulus* and to the quick incarceration of loops of intestine under bands of adhesions; on comparison they will be seen to differ from those of acute tuberculous peritonitis.

(c) *Enteritis* is discriminated from *acute tuberculous peritonitis* by the presence of copious mucous discharges, and by the absence of associated tuberculous lesions, ascites, tumors, and the symptoms of the pronounced typhoid state.

Chronic tuberculous peritonitis often closely simulates *cancerous peritonitis*, owing to the fact that the elongated omental tumor may be met with in both, associated with ascites, abdominal pain, and slight fever. In carcinoma, however, there is an absence of the tuberculous history and lesions, and the presence, sometimes, of a gradually increasing tumor of primary growth, the slowly oncoming intestinal obstruction from pressure, and the cancerous cachexia. Moreover, tuberculous peritonitis occurs more frequently in younger subjects, and is more apt to be interrupted by periods of improvement, followed in turn by rather alarming symptoms.

*Locular exudations* must be distinguished from *ovarian tumors*, and here the history, together with tuberculous lesions elsewhere in the body, the occurrence of febrile attacks, and intestinal disturbance with pain, are of great diagnostic significance. Such cases should be examined by a gynecologist, since, however expert the examiner, when the saccular exudations are located in the pelvic region an exploratory laparotomy must often decide the nature of the condition. Finally, it must not be forgotten that the vast majority of cases of chronic peritonitis are tuberculous.

### TUBERCULOSIS OF THE LIVER.

The liver was formerly overlooked in many instances of tuberculosis, because the lesions, particularly in acute tuberculosis, are often microscopic. In the chronic disseminated variety, however, grosser changes

are observed, the organ being slightly enlarged, pale, and fatty, and presenting an irregular surface like that of an orange. On section, the parenchyma cuts with great resistance, being very dense (tuberculous cirrhosis). Minute gray and larger yellow masses are seen, especially just under the capsule, and small cavities, the result of a breaking down of the cheesy masses and containing pus and bile, are also observed. These changes are most pronounced about the bile-ducts.

**Etiology.**—The liver is implicated in all instances of acute miliary tuberculosis. It is also involved secondarily in chronic tuberculosis of the lungs, pleura, peritoneum, spleen, lymphatics, etc.

**Symptoms.**—This is a common condition, the organ being appreciably enlarged and its surface presenting irregular, palpable prominences. The clinical features of perihepatitis and peritonitis are often found in combination. Ascites may be present, but is rare.

### TUBERCULOSIS OF THE GENITO-URINARY SYSTEM.

(1) **Tuberculosis of the Kidneys.**—This may be primary or secondary, the secondary form being the more common, and it may be either unilateral or bilateral. Infection occurs through the blood in some instances.

**Pathology.**—The process begins in the calices and apices of the pyramids (papillæ), thence proceeding to the pelvis of the kidney, so that early the condition may be pyonephrosis. The morbid changes then extend to the ureters, and sometimes to the bladder and prostate, and instances are even met with in which the process seems to have crept from below upward, starting from the bladder or prostate. The tubercles pass through the usual stages of caseation, necrosis, and suppuration, and destruction of the renal tissue to a greater or lesser degree occurs, with the formation of cysts containing cheesy material in which lime-salts may be deposited. When the process invades the kidneys through the blood, it may be limited largely to the cortical layer and give rise to nodular tuberculosis with caseous masses, yet with little loss of renal substance. There are not a few cases in which the chief lesion is a tuberculous pyelitis. In the latter class the ureters show extensive involvement, such as thickening of the coats with caseation and ulceration of its mucosa; the bladder, deep urethra, and prostate may also be involved. While it is difficult to judge of the relative ages of the lesions in different organs, I cannot escape the conviction that in this group of cases renal tuberculosis is an *ascending* process and follows uretero-cystic tuberculosis. As before stated, however, most instances are *descending*. Although both kidneys are finally involved in most instances, the lesions are usually much more advanced in one kidney than in the other, and hence for a considerable period the disease is probably unilateral.

**Etiology.**—Of disposing factors *age* and *sex* deserve especial mention, most cases occurring during middle life, though they are by no means rare both at an earlier and a later period.

**Sex.**—The disease is much more frequent in males than in females. As stated, the bacilli reach the kidneys with the blood-stream, producing primary renal tuberculosis, but invasion may also take place through the lymphatics or through direct extension from adjacent structures.

**Symptoms.**—In many cases there are either no renal symptoms or



none until a late stage is reached, but the symptoms of pyelitis are usually present. Pyuria may be the only symptom for a long time, and this symptom, according to certain authorities, points directly to cystitis. When the latter condition is present, however, the micturition becomes frequent and there is vesical tenesmus. Pain in the side chiefly affected is complained of, and is sometimes not unlike renal colic; hematuria is not rare; and a cystoscopic examination may show the blood to be of renal origin (Tuffier). It is useful also in showing the state of the bladder-mucosa. The demonstration of tubercle bacilli in the urine, especially if arranged in S-shaped groups, is diagnostic (Frisch). When the bacilli cannot be found, inoculation-experiments upon guinea-pigs and rabbits furnish an accurate criterion, though it must not be forgotten that tubercle bacilli may find their way into the urine from more distant tuberculous foci. Polyuria is sometimes present, as well as albuminuria; the urine may also show tube-casts (rarely) and pus-cells. Macroscopic cheesy masses are occasionally found.

The general features are often marked, but not until the affection becomes advanced, chills, fever of a suppurative type, emaciation, and increasing debility being the chief symptoms. Associated tuberculous lesions, especially of the lungs, are constantly observed.

*Physical Signs.*—Inspection may show a tumor-like prominence on the side chiefly affected, though rarely of large size. Palpation often detects tenderness, and the limits of the organ may be defined by careful firm pressure with the finger-tips.

*Diagnosis.*—It is difficult to discriminate *calculous pyelitis*. In the latter, however, the pain is severer, the tumor-mass larger, and the hemorrhage more frequent than in tuberculous nephritis. The discovery of tubercle bacilli or the demonstration of tuberculosis of the lungs or of other organs would remove all doubt. These two affections may coexist.

(2) *Tuberculosis of the Ureter and Bladder.*—This is almost always secondary to tuberculous disease of the pelvis of the kidney above, or of the deep urethra, testes, or prostate below. When primary, as rarely happens, the bladder is in most instances invaded last. The *symptoms* are those of chronic cystitis, and in all cases in which no other cause for the latter can be found the primary tuberculous lesion must be sought for and the urine carefully examined for bacilli. The usefulness of the tubercle bacillus as a final point in diagnosis is impaired in this situation by the fact of the frequent presence of the smegma bacillus in normal urine. Some observers state that the smegma bacillus can be distinguished by decolorizing with absolute alcohol, which will take place in about two minutes, while with the tubercle bacillus a very much longer time is required. Others say this is not sufficient, and that only their methods of culture-growth or inoculation will distinguish them. With the development of ulcerative lesions hemorrhage is apt to arise.

(3) *Tuberculosis of the Vesiculæ Seminales, Prostate, and Testes.*—The prostate gland and testes are frequently invaded in genito-urinary tuberculosis, and the vesiculæ seminales somewhat less frequently. The morbid process leads to the formation of cheesy nodules, which may, though comparatively rarely, disintegrate, causing excavations or perforation. Rarely, the tubercle does not pass through the stage of caseation, but merely shows the presence of numerous embryonic cells.



**Etiology.**—The condition is usually secondary, but the existence of primary tuberculosis in these organs cannot be denied. Testicular tuberculosis may begin at any period of life, and is of rather frequent occurrence in infants. When it occurs in the latter, it is part of a more general tuberculous infection, and is in many instances undoubtedly congenital. In some cases it may be a late hereditary affection.

**Symptoms.**—In the testicle, tuberculosis, as a rule, induces a painless, protracted orchitis, though when cavernous lesions occur the symptoms are more acute. In prostatic tuberculosis the bladder is highly irritable, there is great distress felt in the thigh and groin, and micturition is very painful. Catheterization, particularly if the urethra (as is very rarely the case) is the seat of tuberculous ulceration, causes most excruciating suffering, and there may be signs of stricture. Rectal palpation detects in the prostate firm nodules varying in size from a pea to a bean, together with enlargement of the organ.

**Diagnosis.**—The diagnosis of tuberculosis of the prostate is easily made from the vesical symptoms, the presence of tuberculosis in other organs, the result of rectal examination, and the detection of bacilli in the urine. Syphilitic involvement of the testicle is sometimes excluded with difficulty; in the latter disease, however, the surface of the swollen organ presents greater irregularities, and is even less painful than in tuberculosis. The absence of the history of syphilitic infection and the presence of tuberculosis in other organs, particularly in the uro-genital system, are valuable points in the discrimination.

#### TUBERCULOSIS OF THE FALLOPIAN TUBES, OVARIES, AND UTERUS.

Tuberculosis of the tubes in women is a not infrequent condition, and may be primary.

**Etiology and Pathology.**—The tubes, as a result of infiltration, are thick, hard, and bound down by false membrane. Their ends are generally closed, but the intervening portion is dilated, and contains mucus, pus, and cheesy material. A catarrhal salpingitis is generally in association. *Uterine tuberculosis* is rare, and its origin is usually attributable to similar involvement of the tubes.

The disease is most common during the period of greatest sexual activity, but young children may suffer (*vide* literature of Hennig), and in them the ovaries and uterus may be implicated without participation of the tubes, as in cases reported by Gusserow. At any period of life the lesions may be microscopic; they usually, however, excite marked local peritonitis, which may become general, with the development of ascites. The process may extend to the vagina.

**Diagnosis.**—The age, family history, and signs of the tuberculous diathesis must be noted. The disease does not distinguish itself from other tubal tumors by anything characteristic on bimanual palpation. Cases occur with ascites and also without, and in the latter variety plaque-like thickening of the subperitoneal tissue is an aid to diagnosis. The uterine secretions should be examined for bacilli in all obscure cases. Ashton advises an exploratory incision or puncture and examination of the contents of the peritoneum or tubes for bacilli.

## TUBERCULOSIS OF THE MAMMARY GLANDS.

This is a rare form of surgical tuberculosis, in which the mammary glands present fistulæ and ulcers, with induration of the organ and retraction of the nipple. The axillary glands are often enlarged. The tuberculous diathesis is usually present, but a positive diagnosis rests crucially upon the finding of the bacilli in the pathologic secretions. A cold tuberculous abscess may occupy the breast.

## TUBERCULOSIS OF THE BRAIN.

**Pathology.**—Tuberculosis of the brain occurs in two forms, one of which, acute tubercular meningitis, has been previously described, while the other is a chronic tuberculous infection, usually localized, of the meninges and cortex, and causing meningo-encephalitis. Very rarely the membranes remain intact. The so-called solitary tubercle is an irregularly round mass, varying in size from a small pea to an apple or even larger. It is generally single, though sometimes there are two, and rarely even three, nodules. The tubercle may be imbedded in, and be contiguous with, the brain-substance, or may be separated from the latter by cysts. The peripheral zone is formed largely of connective tissue, is lighter in color (often translucent), and may contain miliary tubercles, while the central portion, which is cheesy as a rule, may liquefy and thus form a small cavity containing a purulent-looking material. Here, as elsewhere, the tubercles may calcify. They are seen with greatest frequency in the inferior portions of the brain—cerebellum, pons, and medulla—and are rare in the cerebral cortex.

The new growths may compress the longitudinal sinus, inducing thrombosis; they may interfere markedly with the circulation, causing cerebral softening; and, finally, they may excite acute tuberculous meningitis. Tuberculosis of other organs, particularly the lungs, bronchial glands, joints, and bones, is usually found as an associated condition.

**Etiology.**—The disease occurs with especial frequency in young subjects, and, according to the statistics of Pribram, in about three-fourths of the cases before the fifteenth year. The symptom-picture is identical with that of brain-tumor, and hence will be appropriately given under the latter head.

## TUBERCULOSIS OF THE SPINAL CORD.

The lesions are those of solitary tubercle of the brain. It is an extremely rare condition, and almost invariably secondary. (For symptoms, *vide* Spinal Tumor and Meningitis.)

## TUBERCULOSIS OF THE HEART.

In acute miliary tuberculosis gray granulations or larger yellow tubercles may be found throughout the tissues of the heart. More frequently than was formerly supposed does cardiac tuberculosis also result from chronic tuberculosis of adjacent organs. Illustrative cases have been reported by Townsend and Waldeyer. Constantin Paul has in these

cases observed tubercles in the wall of the left auricle, as well as in the infundibulum of the pulmonary artery in several instances.

*Valvular tuberculosis* is, I believe, even more common, though few cases have been reported. Londe and Petit in one instance found the heart much affected, and discovered on the mitral valve several vegetations the size of lentils, which showed tubercle bacilli. I have seen two cases—one in which the mitral, and another in which the tricuspid, valve was affected, associated with latent pulmonary and mediastinal tuberculosis. Doubtless there are cases in which the valve-lesions form the central and most prominent point in the picture.

### TUBERCULOSIS OF THE ARTERIES AND VEINS.

This may arise consequent upon extension of a tuberculous process into the vessel, as in chronic phthisis. It causes infiltration of the arterial wall, resulting in thrombosis, or the vascular tubercles may caseate and soften, thus leading to hemorrhage. In tuberculous meningitis the arterial lesions are conspicuous. The perforation of a vein by an old focus is followed by a distribution to all parts of the body of numerous bacilli and acute miliary tuberculosis. Infection of the arteries may also occur through the blood. In a case of chronic tuberculosis Flexner found a fresh tuberculous growth in the aorta which had no connection with the cheesy masses outside the vessel (Osler).

**General Prognosis.**—The prognosis is best reached as in other infectious diseases—namely, by taking into account (*a*) the severity of the type of the disease; (*b*) the presence or absence of frequently associated diseases or complications; and (*c*) the numerous circumstances connected with individual patients.

(*a*) **The Severity of the Disease.**—Though there are no accurate criteria, we may judge of the severity of the disease by its progress, by the result of proper treatment, and from certain symptoms. If the fever be high, the prostration marked, and the local lesions rapidly advancing, we may safely infer that the disease is of aggravated type. With these certain other considerations are closely connected—the stage of the affection and the extent of the local lesions. Thus at an early stage the prognosis is more hopeful than at a late period, and, similarly, when the lesions are strictly localized at one apex it is more hopeful than when they have reached the stage of extensive cavity-formation or are bilateral. As already stated, a certain proportion of the cases manifest an inherent tendency to spontaneous arrest or even cure, and this may occur even after the stage of excavation has supervened. Notwithstanding this truth, however, it is well to make in all undoubted instances of the disease a guarded prognosis. A common error is the mistaking of a temporary for a permanent arrest of the tuberculous process, and in the natural history of the affection the fact was emphasized that its course was interrupted by periods of comparative comfort and noticeable improvement, followed by sharp exacerbations.

(*b*) **Associated Diseases and Complications.**—These unfavorably modify the prognosis to a greater or lesser degree, the fatal termination often being hastened by chronic nephritis, by gastric complications, and by intestinal and laryngeal involvement. Some of the accidents of the dis-



ease may also precipitate a fatal result (*vide* Modes of Death). The sudden appearance of intercurrent acute pneumonia, whether tuberculous or not, is indicative of danger. Other complications presented by the lungs and other organs have been detailed in the Clinical History.

(c) **Circumstances connected with Individual Patients.**—(1) A feeble, delicate constitution, either acquired or inherent (tuberculous diathesis), increases the gravity of tuberculosis.

(2) When the general symptoms show marked improvement, and especially if the fever subsides and the patient gains flesh and strength, the outlook at once brightens.

(3) **Hygienic Surroundings.**—When the hygienic regimen under which the patient lives is of the best, the prospect is more hopeful than when it is faulty. An improved diet often decidedly aids favorable progress, while a defective one often turns the scales against recovery. Equally influential for good is a pure atmosphere, while, *per contra*, a vitiated one is most injurious.

(4) **Age.**—In young subjects from five to fifteen years of age tuberculosis often pursues an acute course and the mortality-rate is exceedingly high. Chronic tuberculosis may occur, however, less frequently, and under appropriate surroundings may lead to recovery. In chronic phthisis “the younger the patient the shorter the duration.” I have frequently observed that patients who give a history of pleurisy or other phthisical manifestations early in life do not bear chronic phthisis well should it develop at a later period. During old age—a time of life at which tuberculosis is not uncommon—the disease (especially the pulmonary variety) is usually more or less latent, and, owing to coexistent emphysema and chronic bronchitis, pursues a slow course.<sup>1</sup>

(5) The gravity of tuberculosis may be determined with some degree of accuracy by the use of creasote in gradually ascending doses. Hence this agent has a value, not only from a diagnostic but from a prognostic view-point.

#### TREATMENT OF TUBERCULOSIS.

**Prophylaxis.**—(1) This embraces thorough and prompt disinfection of the sputum as the best preventive element. To this end the patient must be taught to expectorate at all times into a spittoon or spit-cup which

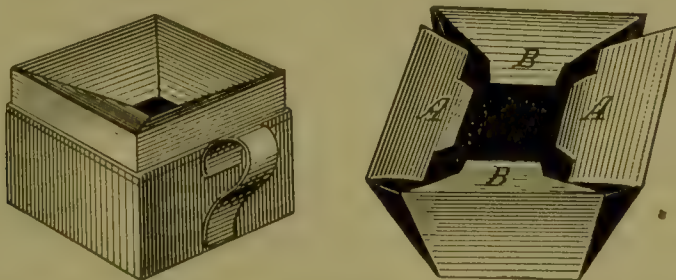


FIG. 30.—Pasteboard spit-cup for receiving infectious sputum. When used the pasteboard can be removed from the steel frame and burned.

contains a proper disinfectant solution, and when the breaking-down stage has arrived portable flasks (*e. g.* Dettwiler's) containing an antiseptic solution must be worn by the patient, even while out of doors. Afterward

<sup>1</sup> A physician should not neglect to examine the sputum in suspicious cases for bacilli.

the sputum is to be destroyed by boiling or burning and the spit-cup sterilized. Paraffin flowers are most desirable for consumptive patients.

(2) **Isolation.**—*Isolation* stands next to disinfection as a means of limiting the spread of tuberculosis, and after the stage of softening is reached the patient should invariably occupy a separate apartment. Phthisis obeys the laws of infectious diseases, and despite great care the room and bed occupied by the consumptive become in time a source of infection through the drying of the expectoration. Hence, unwashable hangings and upholstered furniture, as well as other objects that facilitate the harboring of the bacilli, should be removed from the sick-room. The floor of the apartment should not be carpeted, but may be in part covered with rugs that can be frequently taken up and shaken in the open air. For like reasons, special hospitals for the treatment of the tuberculous poor are a prime necessity. I quite agree with Flick in recommending that tuberculous patients in the infectious stage of the disease should be retired from occupations in which they can infect others, and the pensioning of those who cannot be maintained in hospitals.

(3) **Compulsory registration** of tuberculous (pulmonary) patients is desirable, since it would serve to emphasize the importance of certain sanitary measures both during and after the course of the case.

(4) **Government Inspection of Dairies and Slaughter-houses.**—This is the serious business of the State, and, since infection through food, especially milk, is quite common in infants, skilled veterinary inspection of dairies is of prime importance. Of the greatest benefit would be the killing of all tuberculous cattle, and of less though decided efficacy the confiscation at the abattoirs of all carcasses that present marked lesions.

(5) **The popularizing of information** relating to the dangers of, and the means of stamping out, this great scourge. This may be in part accomplished by mural placards, stating simple, plain facts about the way in which the disease is spreading. Armaingaud suggests the placing in the homes of the people printed matter in a form suitable for preservation.

(6) **The Removal of Known Predisposition to the Disease.**—The tuberculous diathesis, whether inherited or acquired, must be overcome, if at all, by vigorous measures or by better hygienic living. In attempting to remove the phthisical tendency the physician must place chief reliance upon the most favorable environment attainable. The value of a change of residence—from the city to the country, the seaside, or the mountains, according to circumstances in individual cases—cannot be overestimated. It often renders predisposed persons immune. For some, and particularly young subjects, an equable climate (Southern California or Florida), that will enable them to live an out-door life, is to be preferred. Attention to the food must not be forgotten. Milk is excellent and should be used freely. Daily sponging of the neck and thorax with cold water is beneficial, and appropriate light gymnastics should be instituted if the subject be old enough. In-door occupations are to be forbidden, and the ventilation of living- and bed-rooms must be looked after carefully.

Flannels are to be worn next to the skin all the year round, for, while it is needful to lead an out-of-door life, we must seek to avoid catarrhal affections, to which these subjects are very prone, and which, it will be recalled, predispose to active infection.

Tuberculosis is apt to develop especially in children while convalescing



from acute fevers, and hence during this period every precaution against catching cold must be observed, and the child be strengthened speedily by vigorous feeding, pure air, and tonics. In children predisposition often results from obstructions in the nose and from persistently enlarged tonsils; and whenever these conditions exist they should be promptly removed. All local foci of tuberculosis in children—glandular, osseous, and articular—must be attacked surgically.

**Treatment of the Disease.**—The treatment of tuberculosis, regarded as a parasitic disease, presents two leading indications. One has reference to the destruction of the specific cause, the tubercle bacilli, by the use of antiseptic inhalations or of some parasiticide taken internally. Of the numerous substances used by inhalation, few have given satisfactory results, this being largely due to our inability to convey them to the smaller bronchi in a sufficient degree of concentration. They are best adapted to, and most efficacious in, cases in which the larynx is involved.

While the antiseptic treatment, both by inhalation and by means of the introduction into the blood of antiseptic substances, is to be carried out, it accomplishes nothing more than the arrest of the growth and development of the bacilli, and that in an indirect manner. The inhalation of antiseptic substances may be accomplished in various ways—by inhaling vapors, by the use of the steam-atomizer, or by some form of “respiration-inhaler.” I have long employed the Robinson inhaler, the sponge of which is moistened with a few drops of a mixture made of equal parts of creasote, chloroform, and alcohol, the patient wearing the inhaler nearly all the time when not eating or sleeping. Unfortunately, most patients object to the constant use of this instrument. The chief among other antiseptics thus employed are carbolic acid, terebene and terpin hydrate, turpentine, thymol, iodoform, oil of peppermint, and a spray of a solution of sulphurous acid. These agents may be variously combined. The injection of antiseptics into the diseased areas in the lungs, as recommended by Pepper, has been for the greater part abandoned. As pointed out by Osler, however, the remarkable results that surgeons have recently obtained in the treatment of joint-tuberculosis by injections of iodoform point to this as a remedy which will probably prove of service when injected directly into the lungs.

The most common, because least objectionable, mode of introducing this class of substances is by internal administration. According to the results reported from all quarters of the world, creasote thus employed alone enjoys the confidence of the profession; and in common with numerous other observers I have found its continued use to be followed by lessened cough and expectoration, lessened fever, and by a lessening or cessation of the night-sweats, with a gain of strength and weight as the natural consequence. In my own hands its beneficial effects have been manifested at the end of two or three weeks. It must be borne in mind that the dose is to be gradually increased to the point of gastric tolerance, which in my experience usually does not exceed 15 to 20 drops (0.999) three times a day. Larger doses are, however, sometimes tolerated.

Following, in the main, the practice of Trudeau, who has used this drug quite as extensively as any other American physician, after reaching the point of tolerance I gradually reduce the dose to and maintain it at 5 or 6 drops (0.333), three times daily. Among the best vehicles are



hot milk, hot water, and diluted alcohol. Recently I have ordered it in capsules, which the patient himself fills at *the time of using*, and have found it a popular and ready mode of administration. When creasote is not well borne by the stomach and its inhalation is seriously objected to by the patient, it may be given by enema, the dose being 20 to 30 drops (1.332), in peptonized milk or mixed with a little egg-white. It has also been employed hypodermically in a 10 per cent. solution in oil of sweet almonds, the dose of which is 1 dram to  $1\frac{1}{2}$  drams (4.0–6.0). Lastly, it has in rare instances been employed by inunction.

Guaiacol, particularly in the form of the carbonate, has of late been quite extensively employed in place of creasote, of which it is the chief active principle. It may be administered in pill or capsule, the dose being slightly less than that of creasoté. It is well tolerated by the stomach, and is broken up in and reabsorbed from the intestinal canal.

Among other remedies prescribed for their supposed parasiticide effect are arsenic, mercuric chlorid, and alcohol, but they are clearly inferior to creasote in this therapeutic rôle.

*Tuberculin* was at one time supposed to exert a specific influence upon the tuberculous processes, but this view has been largely abandoned. Its chief value, as well as employment at the present day, is as an agent in forming the diagnosis.

I am of the opinion that all antiseptics used internally in this disease have for their chief influence a modification of the soil-conditions on which the growth and multiplication of the bacilli depend. They are, in truth, of great value in fulfilling the second leading indication of treatment, which is to overcome the bodily receptivity for the specific bacillus, or to aid the natural defensive processes in limiting the destructive work of the latter. All forms of tuberculosis, however, may heal spontaneously in any stage, this being especially true of the local varieties so common in children, affecting the lymph-glands, joints, and bones.

A large proportion of pleurisies are tuberculous in nature, and although most sufferers from this disease develop pulmonary tuberculosis later in life, many of them apparently heal without the aid of the physician. This is shown by the old pleuritic lesions that are constantly met with at autopsies in persons dying suddenly of other diseases. Spontaneous recovery is seen oftenest in cases that have not progressed to the stage of cavity-formation. Indeed, in the instances in which vomicæ of considerable size have formed, cicatrization or complete cure is out of the question, though they may become encapsulated (quiescent). The percentage of cases in which encapsulated and obsolete tuberculous lesions have been observed at the post-mortem table in persons dying of all causes differs widely with the statistics of different observers. If we consider the cases that are latent from an early period in life, together with those of all ages after childhood, it is doubtless true that in more than 50 per cent. of the human family the bacilli not only gain entrance into the body, but also effect a lodgement. And, since about 14 per cent. of the deaths from all cases can be ascribed to tuberculosis, it follows that unless the conditions are favorable for the growth and development of the bacilli there is manifested a strong tendency to limitation and healing.

In removing the diathesis medicines are unquestionably of less value than the hygienic treatment, the latter in the widest sense of the term

aiming to reinforce Nature's efforts at spontaneous recovery, and embracing four main elements: (1) Climate; (2) Feeding; (3) Special Remedies; (4) Treatment of Leading Symptoms.

(1) *Climate*.—The all-powerful influence of environment has already been pointed out. There is no discrepancy in the testimony of clinicians as to the importance of pure air as a means of rendering the soil unfavorable for the seed. *Per contra*, patients treated in ill-lighted and ill-ventilated sick-rooms and in hospital wards are allowed to remain under conditions that are favorable to bacillary growth, and hence also to extension of the morbid process. Experience and observation have shown that certain climates, selected with particular reference not only to the stage of the affection, but more particularly to the individual, stand foremost as successful modifying influences of the tissue-soil. In any case of tuberculosis that climate is most suitable in which the patient "feels well, eats well, sleeps well, and gains flesh and strength" (Delafield). Until the patient finds such a climate, or if he finds no single climate to produce these results, he should travel from place to place unless special contraindications (excessive debility, etc.) exist. If active tuberculosis has existed, the stay in a suitable climate should not be less than two full years, and if the patient receive benefit from a high altitude, he should remain permanently.

The climatic requisites for a consumptive are (a) pureness, (b) equability, and (c) abundant sunshine. Less beneficial, though important, are (d) dryness and (e) altitude.

(a) *Pureness*.—This requirement is of paramount importance, and thus is explained the fact that mountain air and that of the virgin forest are so helpful in phthisis. Forests, and particularly pine-groves, favor atmospheric purification, since they generate ozone, which oxidizes the impurities contained in the air.

(b) *Equability* has reference to the absence of rapid variations of temperature. On the whole, a relatively low is better than a high temperature, the former being stimulating, and the latter sedative, in effect. It should be pointed out that forests also greatly favor the quality of equability,<sup>1</sup> both as to temperature and relative humidity. They tend to maintain an almost unvarying degree of moisture in their vicinity, thus minimizing the diurnal variations of temperature—a point that is of far greater importance than the question of seasonal variations. Forests intercept and temper the bleak winds of winter, while by their shade and leaf-surfaces they afford a cooler temperature in summer.

(c) *Abundance of sunshine* is demanded by the consumptive. The advantages of sunshine are obvious from the observations made by Munn<sup>2</sup> in the year 1892, when in Denver there was sunshine in 62 per cent. of the possible hours during which it could occur. A *dry* atmosphere has advantages, but that dryness is not an essential element is shown by the fact that patients often do well at places having comparatively high relative humidity, such as Florida, Southern Georgia, Southern California, and the resorts on the south coast of England. The *rarefied* atmosphere of high altitudes, on account of its stimulating effect upon the respiratory function, aids in producing good results, but the pulmonary changes in-

<sup>1</sup> *House-plants as Sanitary Agents; Sanitary Influence of Forest Growth*, p. 312, by the author.

<sup>2</sup> *Medical News*, Aug. 18, 1894.



duced (enlargement of the air-cells, with augmentation of the size of the chest) make it necessary for patients to remain for the rest of their lives. That it is not an essential factor is shown by the excellent results obtained in the oftentimes purer atmospheres at lower levels.

The essential climatic factors mentioned are found in certain American and European resorts. Of the former, the Adirondack region, Colorado, and New Mexico are especially to be mentioned, combining as they do in winter a uniform cold, much sunshine, and purity of atmosphere. A camp- or tent-life in the open air cannot be too strongly advocated. According to my own experience,<sup>1</sup> the Adirondacks meet the indications best in early cases or in patients who have strength enough to lead an outdoor life, and in whom the breaking-down stage is not too far advanced. Some cases, in the early stage, also do well at Thomasville, Ga., Southern California, and in other mild, equable climates. The latter resorts possess the added advantage of affording an opportunity of gaining a livelihood. Among foreign resorts, Davos possesses about the same advantages as may be met in Colorado, New Mexico, and the Adirondacks, while the resorts in Southern Italy and France are comparable to Southern California, Southern Georgia, Florida, and the Bermudas in this hemisphere. Good culinary and home comforts are considerations of little less importance than the climate.

There is a class of phthisical patients in whom the disease progresses rapidly, with the frequent occurrence of hemoptysis, and who, suffering from the debility of the advanced stage, are precluded from the possibility of travelling long distances or becoming acclimated. Such patients and persons with weak hearts should not be sent to high altitudes, but to a genial, warm climate with a fair degree of saturation. Briefly, the atmosphere of forest resorts possesses certain unmistakable advantages for this group of sufferers. Hence they should be sent into the neighborhood of the nearest forest in a mild latitude where reasonably good food and other comforts of life are obtainable. In this connection the superior value of the highly ozonized and terebinthinized atmosphere of the pine-groves cannot be too strongly emphasized. It is especially serviceable in cases in which the laryngeal or bronchial element is marked.

There is also a class of stay-at-homes made up chiefly of the tuberculous poor. Easily accessible sanatoria should be provided for these by the larger cities, and, if practicable, choice should be made of a well-sheltered locality in the woodland. Such sanatoria would combine the supreme advantages of a uniform temperature and a purified air. Sanatoria for the treatment of pulmonary tuberculosis among the better classes are also needed. The Saranac Sanitarium and Falkenstein, near Frankfurt-on-the-Main, and Görbersdorf, are excellent examples, and their reports show encouraging results. Bowditch<sup>2</sup> has obtained favorable results from the treatment of 51 cases of incipient pulmonary tuberculosis in the sanitarium at Sharon, near Boston. Attention to every hygienic detail was also given. The sanatoria should take the form of cottages and pavilions, and should be officered by an intelligent physician. Solaria, in connection with city hospitals for advanced cases, would, I am certain, yield gratifying results.

Home sanatoria can be readily improvised by stocking living apart-

<sup>1</sup> *Loc cit.*, p. 313.

<sup>2</sup> *Boston Medical and Surgical Journal*, July 12, 1894.



ments, preferably those having a southern exposure, with growing plants. The beneficial influences arising from the presence of the latter are ascribable to two functions—the generation of ozone, and transpiration.<sup>1</sup> Such a pleasant retreat furnishes a uniform degree of moisture in the air, and is especially adapted to the winter season. During the midsummer months the patient should live out of doors or in the balmy air of a neighboring forest. Home sanatoria are to be thought of when the expense of a sojourn at a resort cannot be afforded.

(2) **Feeding.**—The diet should be both nutritious and generous. Too close attention cannot be bestowed upon the feeding and upon the condition of the gastro-intestinal tract. Above all, when the remedies prescribed interfere in the slightest degree with the function of the stomach they must be stopped. Such disturbances most frequently arise from the use of syrupy cough-mixtures, cod-liver oil, large doses of creasote, and less frequently from the prolonged use of bitter tonics.

Such easily assimilable albuminous articles as milk, eggs, and the lighter forms of meat, together with fats, should be taken freely. The hydrocarbons are urgently needed, but they must be taken with care lest they derange the digestive function. The appetite is often poor or even lost in the early stage. When this is the case the patient should keep in the open air or try a brief change of air by going, if it be possible, to the seaside. It is, however, generally needful to resort to systematic feeding, giving a small quantity of nourishment, such as milk, meat-juice, egg-white, and the like, at stated intervals. The French method of forced feeding deserves a trial if there be absolute loathing for food. It consists of first washing out the stomach with cold water, and then introducing the following mixture thrice daily: 1 liter of milk, an egg, and 100 grams of very finely powdered meat. As a rule, the patient cannot be induced to swallow this, and it then must be poured through a stomach-tube. If begun sufficiently early, the method gives truly brilliant results. When the temperature is above 100° F. (37.7° C.) the patient should be kept at rest. In a minority of the cases the appetite is ordinarily keen, often as a result of change of air, and usually pursues a relatively favorable course. The following combination will be found useful in assisting the appetite:

Ry. Sodii bicarb.,	3iiss (6.0);
Tr. nucis vomicæ,	f3iiss (10.0);
Glycerini,	f3ss (15.0);
Inf. cascarillæ,	q. s. ad f3iv (120.0).

Sig. 3ij (8.0) t. i. d., in water, fifteen minutes before mealtime.

Other simple bitters and mineral acids may be tried, and there are many cases in which the judicious use of stimulants, particularly wines and malt liquors, aids the appetite and digestion materially. The chief indications for the exhibition of alcohol are—loss of appetite, feeble digestion, and weak, rapid action of the heart. Occasionally they will tend to aggravate these indications, rather than relieve them, and in this event they should be promptly discontinued. Brandy or whiskey in the form of milk-punch may be given freely in the advanced stage, and more

<sup>1</sup> *Loc. cit.*, p. 168.

especially during the morning hours when the temperature is subnormal. Lavage has helped some of my cases immensely, sometimes curing a complicating gastritis.

(3) **Special Remedies.**—These increase the bodily resistance by improving the chief nutritive processes, but do not directly affect the tuberculous lesions; among them cod-liver oil occupies first place. It is useful in a certain proportion of the cases, in which it seems to have more than a mere food-value. It is, however, exceedingly difficult to estimate the therapeutic importance of the oil while other measures—dietetic, hygienic, etc.—are being brought to bear. It sometimes causes further impairment of the appetite and digestion or sets up intestinal disturbances, and under these circumstances its effects are harmful; on the other hand, when well borne it may be very properly employed. The commencing dose should be small (3j–4.0, once or twice daily, to be increased after a time to 3ij–8.0, two or three times daily). It should be taken about meal-time, but whether before or after may be left to the dictation of experience in individual cases. In most instances it is best tolerated after meals. When the pure oil is not well borne, it may be given in combination with an alkali (lime, soda, etc.). Some patients prefer to take it in connection with stimulants, but this should be advised against as long as it can be administered in other ways. Should the oil be found to disagree after it has been used for a considerable length of time, it should be temporarily discontinued. This often happens during the summer months. As a substitute for cod-liver oil, good cream, preferably Devonshire, may be tried (3ij to 3ss–8.0 to 16.0, three times daily), and if taken regularly cream sometimes gives excellent results.

The *hypophosphites* are especially serviceable in a certain proportion of the cases, though there is some difference of opinion in respect to their therapeutic worth. I am in the habit of employing them in cases in which the oil is not tolerated and in which there is a feebleness of intestinal digestion. The commencing dose should be 1 dram (4.0), increased to 2 (8.0) if it is well borne.

*Arsenic* is warmly advocated for its general influence on this disease, but clinical experience has taught us that it is useful in some cases and not in others. The dose should be small, in order that it may be given for a long period of time without interruption, and it matters little what form is employed. Jacobi speaks highly of arsenic, as well as of *digitalis*, in tuberculosis in children.

The *advent of an acute disease* may arrest and cure a tuberculous process. Thus, the symptoms and signs of advanced tuberculosis have disappeared after an attack of virulent small-pox and acute rheumatism (Harris and Beales). Hysteria also exercises an ameliorating effect upon pulmonary tuberculosis, according to the observations of Gibotteau,<sup>1</sup> who advises against treatment of the former disease in tuberculous persons.

(4) **Treatment of Leading Symptoms.**—Certain symptoms demand attention: (a) *Cough.*—This is often quite annoying, sometimes interfering with sleep, eating, and digestion, and even inducing vomiting; but it is to be remembered that cough is an essential feature of the disease, and does not claim attention unless it interferes with the above-named functions. The special cause or causes of the coughing should be determined before any

<sup>1</sup> *The Practitioner*, October, 1894.



attempt is made to treat it. It may be attributable to catarrhal irritation of the upper air-passages or throat, when it is best treated by topical applications, which may be made with the applicator, or in the form of sprays and inhalations. The following substances may be inhaled: compound tincture of benzoin, combined with paregoric or carbolic acid; creasote, alcohol, and chloroform, in equal parts. For local applications by means of the spray sedatives and narcotics should be preferred, and a solution of cocain is sometimes most efficient. The cause may be found in pleurisy or pleuritic adhesions, and for this condition counter-irritants, as iodin, sinapisms, etc., may be used. Pleuritic coughs often demand codein or even morphin in moderate-sized doses. The cough is in most instances occasioned by the tuberculous bronchitis, and to a lesser extent by the vomicae. Cough-mixtures are usually prescribed to meet these indications, but as usually formulated they are apt to disorder the digestive function, and in so far as they have this effect they are positively harmful. Since the relief obtained from their use is due to the sedatives and narcotics which they contain, syrups should be omitted from the composition. I have come to rely upon creasote by inhalation as the remedy *par excellence* for tuberculous bronchitis, and combine it with spirits of chloroform and alcohol. To meet the same indication, and particularly when expectoration is copious, preparations of terebene, terpin hydrate, and tar may be resorted to; and when the cough becomes sufficiently distressing to urgently demand relief, I employ codein (gr.  $\frac{1}{8}-\frac{1}{4}$ —0.008 to 0.016, every three or four hours) in the form of a granule. In the latter stages morphin is allowable, since it is at this time that constant coughing or severe paroxysms of cough, if not restrained, lead to utter exhaustion. Stimulant expectorants may be needful, and when this is the case ammonium carbonate in the infusion of wild-cherry bark is perhaps most efficacious and least apt to disorder the digestion: a few drops of the deodorized tincture of opium or spirits of chloroform may be added.

(b) *Fever*.—Creasote has found a new field of usefulness in the treatment of the fever of tuberculosis. In my experience, at all events, the cases in which it has been used, as above indicated, have shown a greatly diminished febrile movement. Cold or tepid spongings of the body at intervals of one, two, or three hours, according to the intensity of the fever, should be tried. Internal antipyretics are rarely advisable, since during the period of high temperature the cardiac action is much enfeebled; but if urgently called for, the following may be employed: acetanilid (dose gr. ij-ijj—0.129-0.194), phenacetin (gr. iij-v—0.194-0.324). These are to be administered about two hours before the commencement of the daily rise in temperature, and repeated every three or four hours if necessary. Other antipyretics worthy of trial are the mineral acids and zinc oxid, but not quinin, which has utterly failed in my hands. Keeping the patient at rest when the temperature is above 101° F. (38.3° C.) is good practice, though he should be wheeled into the fresh air for as long a time as possible during the day.

(c) *The Night-sweats*.—Among remedies that control the sweats most successfully may be mentioned—atropin (gr.  $\frac{1}{120}-\frac{1}{60}$ —0.0005-0.001); zinc oxid (gr. ij-v—0.129-0.324); sulphuric or gallic acid; muscarin (mij-vj—0.399 of a 1 per cent. solution); agaricin (gr.  $\frac{1}{8}-\frac{1}{4}$ —0.008-0.016). Sponging with equal parts of alcohol and tincture of bella-



donna is very effective, but my own best results have been derived from the use of atropin (gr.  $\frac{1}{120}$ — $\frac{1}{90}$ —0.0005—0.0007) in combination with agaricin ( $\frac{1}{8}$ —0.008).

(d) *Dysphagia* may be a troublesome symptom, especially from involvement of the larynx, and it is best met by local applications of a solution of cocain in glycerin and water (gr. x to  $\mathfrak{z}$ j—0.648 to 32.0), thrice daily before meals. In advanced cases I have resorted to hypodermic injections of morphin (gr.  $\frac{1}{8}$ —0.008) before meal-time.

(e) *Gastro-intestinal Disturbances*.—In nearly all cases of phthisis dyspeptic symptoms and diarrhea come on sooner or later, and for this gastric disorder nothing is so important as a proper regulation of the diet. Perhaps the medical treatment of the gastric symptoms has been dealt with at sufficient length, save that of vomiting, which may come on after meals and constitute a distressing concomitant. Those remedies giving the best results may be adduced as follows: cerium oxalate (gr. v—viij—0.324—0.518), in capsules before meals; calomel and soda in fractional doses; hydrocyanic acid ( $\mathfrak{m}$ ij—iij—0.133—0.199); and chipped ice with brandy sprinkled over it, taken at short intervals, but especially shortly before meal-time.

(f) *Diarrhea*.—The most important factor in the treatment of this symptom is a properly restricted dietary. The medical measures that have been and are employed are very numerous, but it will be sufficient to mention only the most useful, which may be used singly or combined in various ways: bismuth (in large doses), acetate of lead, opium, thymol, salol, benzo-naphtol, naphthalin, etc. To the foregoing may be added two formulæ:

R <sub>y</sub> . Bismuthi salicylat.,	$\mathfrak{z}$ j (4.0);
Pulv. ipecac. et opii,	$\mathfrak{z}$ ss (2.0).
M. et ft. capsules No. xxiv.	

Sig. Two every four hours.

The following acid diarrhea-mixture is excellent; each dose containing—

R <sub>y</sub> . Acid. acetici dil.,	$\mathfrak{m}$ x (0.666);
Morphinæ acetat.,	gr. $\frac{1}{8}$ (0.008);
Plumbi acetat.,	gr. j—ij (0.0648—0.1296).

Complications when they arise must be dealt with according to accepted therapeutic principles.

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## SYPHILIS.

**Definition.**—A chronic infectious disease, due either to inoculation or direct contact with a specific virus. It may either be (1) acquired or (2) congenital, and is characterized by three stages, each of which presents special clinical symptoms and pathologic lesions. The congenital form of the disease is transmitted at the time of procreation either by the sperm virile or by the ovum.

**General Pathology.**—(a) **Primary Lesion or Chancre.**—This appears at the site of inoculation, and is characterized by infiltration of the connective tissue chiefly with round cells, and also by larger epithelioid and giant cells. This is followed by a shallow ulcer of the size of a split pea or larger, of which the base is quite hard, the sclerosis being due partly to cellular infiltration and partly to a marked thickening of the intima of the small arteries (acute obliterative arteritis). Adjacent lymphadenitis, more or less marked, is constantly present, and, rarely, all the peripheral lymph-glands in the body are swollen.

(b) **Secondary Lesions.**—*Condylomata* are the most common. Their favorite sites are the points at which the mucous membrane and the skin are continuous (mouth, anus, etc.); their contour is more or less rounded or oval; their surface readily abraded and usually ulcerated; and their size varies from a pin's head to an inch or more in diameter. Like the Hunterian chancre, their periphery is more or less indurated. Secondary lesions also include skin-eruptions and ocular inflammations, etc., but these can only be referred to under later symptomatology.

(c) **Tertiary Lesions.**—These are circumscribed inflammatory products (gummata) appearing in the connective tissue, bones, periosteum ("nodes"), skin, muscles, brain, liver, lungs, kidneys, heart, testes, etc. The gummata, though usually sharply circumscribed, may take the form of diffuse infiltrations of the affected parts, and vary greatly in size—from a pin's point to a hen's egg. Usually firm, they may be soft, and, particularly on mucous membranes, tend to disintegrate, forming ulcers. Their color is grayish, and on section they show a caseous semi-opaque center, with a fibrous and more or less translucent periphery. They may occur singly or in groups.

Microscopically, the gummata consist of small round cells. The mass thus formed may either be absorbed or persist without change for a considerable period; but in most instances coagulation-necrosis occurs in the center, due to local anemia, and there is a conversion of the peripheral zone into fibrous tissue. The gummata are enveloped in granulations and connective tissue, which contracts, forming cicatrices that often contain calcareous masses on their interior. The lesions of certain structures (skin, mucous membranes, bones, and cartilages) often lead to extensive destructive ulceration and sloughing, and these ulcers as they heal also form typical cicatrices.

**General Etiology.**—**Bacteriology.**—Lustgarten in 1884 described the bacillus of syphilis, which closely simulates that of tuberculosis and the smegma bacillus. It occurs in rod-shaped or curved forms with slightly enlarged ends, and is from 3 to  $5\mu$  long. From the smegma bacillus it is distinguishable by the carbol-fuchsin test; though demonstrable with difficulty in the hard chancre and in secondary lesions, its pathogenic potency is still very doubtful. Inoculation experiments upon lower animals are not possible, since man alone is susceptible.

**Predisposing Causes.**—Since acquired syphilis originates only by inoculation, it is obvious that a break in the cutaneous or mucous surfaces is essential to infection, such as a slight abrasion, fissure, or laceration, etc., particularly of the genital mucosæ. Other surfaces may also be the seat of infection, as the lips, hands, etc.

Susceptibility to the virus is universal, and no age is exempt. Reinfection is exceedingly rare, but does occur; J. William White, however, states that we have easy access to more than one hundred published cases of the reinfection of syphilis.

**Modes of Infection.**—(1) In a great proportion of the cases syphilis is transferred by illicit sexual intercourse, there being, however, other modes of transmission.

(2) *Accidental Inoculation.*—This is not uncommon. (a) Most frequently it is accomplished through the pernicious custom of indiscriminate kissing (lip-chancres), and I have personal knowledge of not less than 8 instances in which infection has occurred through labial contact. Downie reports 4 cases that occurred in children.

(b) The site of inoculation may also be the mouth and tonsils, the virus being conveyed during the low practices of sexual perverts. The wet-nurse may infect the mouths of suckling babes, or, *vice versâ*, the infant may infect the nipple of the nurse.

(c) The obstetric finger may become infected, and Osler mentions one such instance of infection of the back of the hand. Three instances of the sort have come under my own observation, and Fournier gives the details of 40 cases of primary syphilitic infection of the hand. In 30 of these the malady was acquired in medical practice (4 obstetricians, 20 general practitioners, 3 students, and 3 midwives). The remaining cases were attributable to contact and biting.

(d) Humanized vaccine virus may transmit the disease, but this is a rare occurrence (*vide* Vaccination, p. 230).

(e) Accidental infection has, though very rarely, taken place in a variety of other ways—*e. g.* handling foul rags from the hospital ward, by bed-clothing, drinking-cups, the pipe, and cigar.

Kraft-Ebing found that out of a totality of 3455 cases treated at the University of Christiana during twenty-five years (from 1867 to 1894),  $15\frac{6}{10}$  per cent. were of extra-genital origin. The seat of the lesion was upon the lips in 51 per cent., in the throat in 20 per cent., and upon the mammary gland in 20 per cent.

(3) *Hereditary Transmission.*—Paternal transmission (through the semen) is much more common than is maternal, the period of greatest danger being immediately after the father has become infected or during the time of the secondary manifestations. The first-born, if the father be syphilitic, is apt to show well-marked lesions. Appropriate treatment of a syphilitic parent lessens the danger of transmission very materially, however, and in such instances there is little tendency to transmission shown after the third year. On the other hand, a syphilitic father may beget healthy offspring. Syphilitic children are also common to infected women. In the majority of instances of hereditary transmission, however, both parents are syphilitic, and under these circumstances the liability to infect the offspring is much augmented. A woman who has become infected after conception may bear a syphilitic child; though the latter may, on the other hand, escape infection, and particularly if the mother has been actively treated.

Allusion may here be made to Colles's law—that a woman who bears a syphilitic child enjoys, owing to a sort of protective vaccination with the specific virus, perfect immunity, and this in the absence of all signs



of the affection. Coutts<sup>1</sup> does not believe that a mother may absorb from a syphilitic fetus an antitoxin of syphilis which may render her practically immune to the disease.

**Clinical History of Acquired Syphilis.**—(a) **Primary Stage.**—The typical initial lesion (chancre) appears about three weeks after infection, and is followed soon by swelling and induration of the nearest lymphatic glands. The primary sore begins as a red papule, which rapidly reaches its maximum, and then undergoes a central necrosis with the formation of a small ulcer. The adjacent structures become hard or cartilage-like—a characteristic to which the lesion owes its name of “hard chancre.” Secondary suppuration, either of the primary sore or of the lymph-glands in the vicinity, may take place. A small chancre may often escape detection, especially if it be situated inside the meatus. When situated upon a mucous membrane it is always a *chancreous erosion*, which may be so mild and of such brief existence as to come and go without the knowledge of its bearer. Particularly is this the case in the female. The general symptoms are negative in this stage.

(b) **Secondary Stage.**—This is announced about six weeks after the appearance of the infecting chancre by moderate fever (101°–104° F. —38.3°–40° C.), accompanied by languor, headache, bone-pains, impaired digestion, and a slight degree of prostration. The patient, as a general rule, shows signs of anemia (syphilitic cachexia), and angina, with hyperemia of the fauces and hard palate, now appears. There may be minute elevated white patches upon the pharynx; the tonsils may enlarge, and on both shallow ulcers presenting a grayish sharply-defined border appear. They neither excite pain nor spread.

There is also an eruption, which is at first usually *roseolar* and widely distributed, coming out abundantly upon the trunk (especially the chest), buttocks, thighs, and forehead. Another early eruption is the *papular*. The papules are small, hard, and do not ulcerate, while their favorite seats are the scalp, chest, and dorsum of the tongue. The distribution of these early syphilids is symmetric; their outlines are rounded; their color like that of a slice of raw ham (“coppery”); and, as a rule, they excite neither pain nor itching.

Other and later-appearing eruptions may be *squamous*, *vesiculo-papular*, *pustular*, and *tubercular*. These show a tendency to bunch in certain areas, and hence are less diffuse than the afore-mentioned eruptions. Several sub-varieties, however, may appear simultaneously.

The visible mucous membranes (angles of the mouth, tongue, gums, pharynx, vulva, vagina, penis, and around the anus) and the skin may show painful condylomata or ulcers, and especially is this the case in the mouth, where they often stubbornly resist treatment. Recurrences at varying and ever-decreasing intervals are common.

Other frequent symptomatic conditions arise during this secondary period, such as iritis, laryngitis (frequently), choroiditis, retinitis, epididymitis (more rarely), and alopecia. The hairs of the eyelids and eyebrows may fall off and the finger-nails become brittle.

The *secondary* symptoms last from two to three months (the usual duration) to a year or more, and are followed by a period of apparent

<sup>1</sup> “Hunterian Lectures,” *Lancet*, 1896, No. 3789.

good health lasting for an exceedingly variable interval (from a few months to many years) before the tertiary stage sets in. During the secondary stage the symptoms may be severe, mild, or even absent. The severity of an attack of syphilis depends upon the *dose* of infecting virus on the one hand, and upon the condition (both local and general) of the vital functions on the other. This fact explains why a single organ or system, as the brain and cord, is attacked in one instance and some other organ or system in another, and the effect of traumatism in determining the topography of periosteal "nodes" is a good example.

(c) **Tertiary Stage.**—As I have already stated, the secondary period is generally followed by a variable interval of freedom from symptoms, but to this rule there are numerous exceptions, and among not uncommon occurrences may be witnessed the appearance of tertiary symptoms during the secondary stage. Belonging to the third stage are certain skin-eruptions, especially the characteristic *rupia*, which first appears in the form of pustules that break and form ulcers that are covered with dry, laminated crusts "like an oyster-shell." To this stage also belongs *psoriasis*, especially of the hands and feet. *Pustules (tubercular)* which do not scale over also appear. These eruptions involve the true skin, and in healing leave scars, but, unlike the secondary cutaneous lesions, they are neither infectious nor contagious, are not, as a rule, symmetric, and are more liable to be attended by itching. True gummata may develop in the skin and subcutaneous tissue, and these break down and form kidney-shaped ulcers which tend to spread in a serpiginous manner. On healing (a process that is accomplished with difficulty), scars result. Gummata may occur in the mucous membranes, and pass through the stages of ulceration and cicatrization. When situated in the larynx or trachea their healing is attended with narrowing of the organ, and when in the lower bowel or the rectum dysenteric symptoms, followed by actual stenosis, may result.

In the *muscles* gummata occur and form small hard tumors. They may also cause periostitis and death of the bones, especially of the nose, palate, and skull; "nodes" are thus formed, which are situated chiefly upon the tibia and the skull in larger or smaller numbers, and also, though less frequently, upon other bones. These are exceedingly painful, particularly at night, and are very tender under the pressing finger. They may be true gummata, but more often, if not absorbed, they either become ossified or undergo fibroid change, while in rarer cases they suppurate. Chronic enlargement of the lymphatics and of the testicle, with little tendency to suppuration, may be noticed. The pregnant female is apt to abort or miscarry, either as the result of the action of the syphilitic virus upon the ovum or of the presence of gummatous growths in the placenta.

Gummata also occur in the internal organs (*visceral syphilis*), and of the latter I shall speak presently, taking up separately some of the various organs and systems of the body. *Amyloid degeneration* is frequently caused by the acquired form, particularly syphilis of the rectum in women, but very rarely by the congenital.

**Malignant Syphilis.**—By this term is meant a virulent and a fatal form of the malady, which is fortunately rare. The various stages manifest themselves early, and especially the tertiary, as on the forty-



fifth day in a case of Mauriac. The course is rapid and the condition resists all forms of treatment. Roussel narrates a case in which death occurred about one year after the commencement of the disease.

**Clinical Symptoms of Congenital Syphilis.**—These may, though rarely, be identical with those of acquired syphilis, if we except the chancre.<sup>1</sup> Occasionally the characteristic symptoms are present at birth. On the other hand, in the vast majority of instances they appear between the first and fourth months of life (*infra*). The symptoms of inherited syphilis may be grouped according to the time of appearance:

(1) **In the New-born.**—There is a lack of physical development. The babe may be greatly emaciated, it has snuffles, and singultus occasionally sets in soon after birth. Skin-eruptions are rare, except *pemphigus neonatorum*, which appears as bullæ on the palms and soles; among exceptional cutaneous phenomena are gummata around the radio-carpal articulations, palmar psoriasis, and a fleeting roseola. Ulcers and fissures (*rhagades*) may be noticed around the outlets of the body (mouth, anus, etc.); the osseous system may show hyperostoses of the long bones; and the liver and spleen are enlarged. Comby reports 8 cases of pseudo-paralysis due to syphilis in the new-born.

(2) **Early Post-natal Symptoms.**—Most subjects of syphilis hereditaria are born plump and without taint. Romiceano<sup>2</sup> gives the results of his observations of 723 cases of infantile syphilis in which the disease appeared chiefly between the first and fifth months, and only 27 times in all after the sixth month. Rogers's statistics show that among 249 cases, 217 showed symptoms before the end of the third month. The first symptom is generally coryza (syphilitic rhinitis), which is betrayed by a sero-purulent or bloody discharge and a peculiar form of obstructed breathing (snuffles), rendering nursing difficult. The coryza may in some cases be preceded by singultus lasting ten or twenty days (Carini), and ulcers may form in the nose, leading to necrosis of the bones and producing at last a sunken and deformed nose which is highly significant. The coryza may extend to the middle ear and cause otitis media, with deafness and otorrhea as the chief symptoms. The skull may approach the natiform in shape, and the signs of diaphyso-epiphysal inflammation develop.

The *cutaneous symptoms* appear early. The skin has a tawny hue, and an erythematous eruption of the nates and genitals is frequently seen; this is patchy, with well-defined margins, and has the characteristic coppery color. In the same localities papules may appear, while pemphigus may attack the palms and soles. Syphilitic onychia may be present, and the lips and angles of the mouth often show fissures that are of real diagnostic worth. Other symptoms are ulcerations of the skin and mucous surfaces, falling of the hair, and a moderate glandular enlargement.

*Enlargement of the spleen* is a frequent characteristic symptom, and White says that the enlargement of the organ when "painless, subacute, persistent, often preceding the eruptions, should be included in the list of significant symptoms."

Swelling of the liver may also be present, but is of little diagnostic import. Syphilitic infants occasionally manifest a hemorrhagic tendency.

<sup>1</sup> With prenatal syphilis we are not concerned.

<sup>2</sup> *La Progrès médicale*, Paris.



At birth bleeding from the umbilicus may occur; later, into the subcutaneous tissue and from the mucous membranes (gastro-intestinal, vaginal, nasal, etc.). As pointed out by Osler, these cases must not be confounded with Winckel's disease—an acute infectious hemoglobinuria of the new-born.

Among *nervous symptoms*, restlessness, sleeplessness, and a harsh, shrill cry which may be almost constant for days together and due most probably to darting pains, are the chief. Anemia and other evidences of syphilitic cachexia soon supervene.

(3) **Late Symptoms.**—The symptoms of syphilis hereditaria tarda may be arranged in groups (Fournier):

(1) *Those Indicated by the General Appearance.*—There is a retarded general development, as shown by the small stature, undeveloped muscles, the graceful form, and infantile appearance at ages varying from four to twelve or more years. The skin has an earthen tint, and the hair may be scanty and late in its appearance on the face and genitals.

(2) *Skin-cicatrices.*—Cutaneous scars, particularly if multiple and extending over a circumscribed area, are important diagnostic signs. Their form is usually round or serpiginous, and their chief location the mouth, nose, soft palate, and lumbo-gluteal regions.

(3) *Lesions of the Skeleton.*—The natiform skull, “with a transverse enlargement, lateral bulgings, and the flattening in the middle,” is almost pathognomonic. Asymmetric and hydrocephalic skulls are also to be considered, in many cases, as signs of hereditary syphilis, as is a sunken and deformed nose. The thickened, “sabre-shaped” tibia, due to gummatous periostitis, is capital evidence of the disease, while the chicken-breasted thorax is significant.

(4) The *testicles* show an arrest in development (infantile testicles). This is a sclerotic atrophy.

(5) *Hutchinson's triad*, under which title come (a) the Hutchinson teeth; (b) ear-conditions; and (c) affections of the eye.

(a) *The Hutchinson Teeth.*—The teeth may be late in appearing, and the dental arch may be malformed, the teeth presenting various irregularities in form and condition (dental dystrophy).

The incisors, especially the superior median of the second dentition, are notched, and show a thinness of the free edge, an atrophy of the summit, and crescent-shaped erosions. The latter are truly pathognomonic (Fournier).

(b) *Ear-conditions.*—Otorrhea, secondary to naso-pharyngeal catarrh, has already been mentioned, and, in addition, at or about the time of puberty an incurable form of deafness may develop speedily, without the presence of pathologic lesions to explain the same.

(c) *Affections of the Eye.*—These are interstitial keratitis and iritis, affecting both eyes successively.

## VISCERAL SYPHILIS.

**Syphilis of the Brain and Cord.—Pathology.**—The most characteristic and not infrequent lesions are the syphilitic new-growths. Their size varies from that of a bean to that of a chestnut, and they present irregular contours. They are usually situated either in the cerebral hemispheres or on the pons, and rather superficially, connecting directly or indirectly with the dura or pia mater. They may not infrequently originate in the dura mater. In gummata of average size a cut-section shows caseation in spots which are connected and surrounded by firm, translucent, gray or reddish-gray, fibrous tissue; and, according to Gowers, the more irregular surfaces and the irregular caseation serve as important distinctions from tuberculous tumors. When, as is usual, the gummata touch the membranes, meningitis—subacute or chronic, with much thickening—is combined.

As I have said, the condition may begin as a gummatous meningitis, while in fewer instances it may start as a gummatous arteritis. On the other hand, a gumma may secondarily involve a blood-vessel for a considerable distance, weakening its walls, with resulting rupture and intracranial hemorrhage; or it may bring about cerebral thrombosis with secondary softening.

Histologically “the cerebral gumma differs from other similar bodies chiefly in the presence of very large spider-like cells containing an exaggerated nucleus and a granular protoplasm which extends into the multiple, branching, rigid prolongations” (Wood). The arteries, particularly those of the base, may show syphilitic sclerosis; this renders them thick, hard, opaque-whitish, until their lumen is well-nigh obliterated.

Gummatous growths may attack the cord. In a case recently reported by Osler a new-growth occupied the cord opposite the root of the third cervical nerve, “and there were gummata in the cauda equina.” The other gross changes found in connection with cerebral gummata and their secondary lesions (softening, collateral inflammation, etc.) are also observed in syphilis of the cord.

**Etiology.**—Cerebral syphilis is usually a late (tertiary) manifestation—appearing from one to thirty years after primary infection. In some recently recorded cases by Lydston and others nervous lesions became evident during the secondary stage of syphilis, even as early as three months after initial infection. It oftenest develops in cases in which the secondary symptoms have been slight, and may occur in those in which both primary and secondary manifestations have been entirely overlooked. Inherited syphilis affects the nervous system less frequently than does the acquired form, but cerebral gummata have been noted at all periods from the time of birth until after puberty.

**Symptomatology.**—*Imbecility* and *idiocy* may be due to inherited syphilis, but they are probably too often attributed to this cause. The other features simulate those of the acquired form.

*The symptoms of the acquired form* are with few exceptions referable to three affections: (a) epilepsy, (b) brain-tumor, and (c) paralysis.

(a) **Epilepsy** coming on after the twenty-fifth year is usually due to the ravages of syphilis, and a careful search for traces of scars and

bone-lesions, etc. should be instituted. The appearance of the disease may be preceded by psychic disturbance, headache, dizziness, and loss of memory. Hysteric manifestations may also be presented, being probably provoked by the specific lesions. On the other hand, a protracted torpor which may last for a few days or as many weeks may develop. In one of my own cases periods of marked mental excitement, that persisted for three or four days, alternated with periods of almost complete insensibility of about equal duration.

(b) **Brain-tumor.**—The symptoms pointing to brain-tumor will be discussed under this head in the section on Nervous Diseases. The syphilitic nature of the cerebral growth cannot be determined with any degree of certainty except in the presence of a clear history of syphilis—congenital or acquired—and the characteristic symptoms or traces of the primary, secondary, or tertiary lesions. In such cases the diagnosis is almost undoubted.

It must be remembered that the secondaries are either sometimes absent or go unnoticed, and if the patient has had a primary sore, the presence of the characteristic symptoms of brain-tumor (headache, optic neuritis, convulsions, etc.) make the existence of specific nerve-lesions highly probable. The chancre may also be overlooked or denied, and it is in such instances as the latter that the occurrence of convulsions in persons over thirty should excite suspicion, and lead to a trial of the antisyphilitic treatment for further confirmation.

(c) **Paralysis.**—This may take the form of hemiplegia (due to cerebral hemorrhage or tumor) or of general paralysis (*dementia paralytica*). The relation that these affections bear to syphilis will be indicated in its appropriate place in this work in the description of Nervous Diseases. The fact may here be pointed out that syphilis may induce precisely the same changes met with in general paralysis of the insane.

The history of syphilitic infection, together with symptoms of an *atypical* type of spinal tumor, points to *gumma* of the cord. Syphilitic myelitis usually develops within five years after infection, and may pursue an acute or subacute course, though oftener it takes the form of chronic myelitis. The latter attacks by preference the lumbo-dorsal section of the cord—a fact corroborated by the character of the symptoms. The clinical features, however, are not distinctively syphilitic; neither does the effect of treatment in the slow sclerotic form add fresh light as to the specific nature of the trouble, since the process is uninfluenced by the most vigorous antisyphilitic measures. When the etiologic influence of syphilis can be shown, especially in the absence of other causes, the diagnosis of syphilitic myelitis rests upon more certain ground. Acute syphilitic myelitis gives an unfavorable prognosis. The relation between syphilis and spinal meningitis, primary spastic paraplegia, and locomotor ataxia will be more fully dwelt upon under the latter affections.

### SYPHILIS OF THE LIVER.

In my experience the liver, with comparative frequency, bears the stress of visceral syphilis.

**Pathology.**—The lesions may be thus classified: (a) **Diffuse Syphilitic Hepatitis.**—This is met with chiefly in congenital cases, though I



have seen an instance in an adult who died of cerebral hemorrhage, the occurrence of which in adult life has been questioned by some. The liver is uniformly enlarged, firm, and resists the cutting knife. Its color is grayish-yellow.

The microscope shows a marked increase in the connective tissue and a cell-infiltration throughout. From intense, focal cellular infiltration miliary gummata may result; these undergo contraction, diminishing somewhat the size and altering the shape of the organ.

(b) **Gummata.**—These may be seen in congenital cases (chiefly the miliary gummata). As seen in the adult, hepatic gummata are disseminated nodules, with the usual central, cheesy mass surrounded by a zone of grayish fibrous tissue and varying in size from a hazelnut to an apple. They form separate tumors, whose favorite seat is the convex surface of the organ, especially near to the suspensory ligament. They are usually tertiary lesions, and do not appear until a number of years (two, three, or four) after infection. These so-called syphilomata in the advanced stage contract, and the liver will be found smaller than the normal. Deep furrows, due to contracting fibrous bands traverse the organ in different directions and divide it into lobes of various dimensions. Gummata frequently undergo fibroid change, but more rarely they soften and liquefy (Wilks). On the other hand, before contraction occurs the liver is increased in size and the gummata form protuberances on its surface.

(c) **Gummatous Arteritis.**—Briefly, this may affect both the portal vein and hepatic artery, though syphilitic endarteritis is situated chiefly in the smaller branches of the latter.

(d) **Perihepatitis.**—Here Glisson's capsule is thickened, owing to augmentation of its connective-tissue elements. From the latter there dip into the hepatic tissue cicatricial bands, particularly along the portal canals, which may change somewhat the shape of the organ. Section shows admirably the pale scar-like tissue.

**Clinical History.**—The affection may exist without symptoms. In the congenital form, however, we have signs of hepatic enlargement, with icterus, the spleen being likewise large and firm, as a rule. The history and associated lesions are necessary to a certain diagnosis.

In the *adult* syphilis of the liver does not usually attract attention until the gummata interfere with the portal circulation. As they undergo contraction they tend to occlude some of the portal branches, or they may, on account of their situation, exert pressure upon the vena porta itself. In either event the evidences (ascites and splenic enlargement) of portal obstruction will develop as in alcoholic cirrhosis. The gastro-intestinal symptoms common to the latter disorder are also present, and obstructive jaundice may supervene, though it is, comparatively speaking, rare. Pain, usually localized to some particular spot over the right hypochondrium, is sometimes complained of, and may be quite severe, while pressure over the painful area elicits great tenderness.

**Physical Examination.**—In the early stage, while the organ is enlarged, flattened, irregular protuberances may be detected by the palpating fingers. At a more advanced period ascites may interfere with palpation, and in such cases an aspiration of the fluid will enable one

to feel the syphilomata. Finally, in the stage of contraction the results of palpation are obviously negative.

There is a group of cases in which the clinical picture is that of advanced amyloid disease of the viscera. The liver and spleen are enlarged, the urine is increased in amount and contains albumin and tube-casts, and finally dropsy supervenes. Here *secondary amyloid* degeneration has occurred.

**Diagnosis.**—This rests upon the etiology, the presence of scars in the throat or on the skin-surface, bone-lesions (especially irregularities of the tibial surfaces), or other evidences of the ravages of the disease, and upon moderately good general health. The causal factors of ordinary cirrhosis, alcohol, etc., must be carefully excluded. The most important symptoms are the hemispheric prominences (sometimes separate) on the surface of the liver, and the localized pain.

The grouping of symptoms in this disease bears a close resemblance to those of cancer of the organ, but there are points of dissimilarity which I have contrasted in the subjoined table:

SYPHILIS OF THE LIVER.	CANCER.
History of heredity or of infection.	History of heredity or of primary growth.
Occurs congenitally, or, if acquired, at any age.	Never congenital. Usually occurs after the age of forty.
Often accompanied by symptoms of tertiary syphilis—alopecia, rupia, syphilitic iritis, etc.	Often preceded by the primary growth in pylorus, uterus, mammary gland, etc.
Jaundice and ascites are common, especially the latter. No cachexia.	Jaundice and ascites are rare. Marked cachexia.
The margin, on palpation, is markedly irregular, and neither nodular nor umbilicated.	Often the margin reveals the presence of umbilicated nodules.
Recovery may follow, or the affection may last for years.	Always fatal. Duration usually from a few months to a year.

The **course** and the results of antisyphilitic treatment are of value from a diagnostic view-point. The course is slow and often interrupted temporarily by improvement, if not arrest of the disease, while appropriate treatment sometimes leads to recovery, with a complete disappearance of the tumor-mass, as occurred in two cases of my own.

#### SYPHILIS OF THE ALIMENTARY TRACT.

The lesions in the mouth have been for the most part considered. In the tongue gummata often develop. A decidedly fissured appearance of the organ and whitish scar-like patches upon the surface may be observed in syphilis, but have no essential connection with that disease. Gummata also appear on the posterior wall of the pharynx and lead to ulceration, which may cause fatal hemorrhage by erosion of adjacent large blood-vessels (internal carotid, etc.). The walls of the esophagus may also be invaded, resulting usually in stenosis.

The stomach-walls may be infiltrated, though they are rarely ulcerated; syphilitic ulcers, however, may appear in the intestines, and the condition may lead to perforation and peritonitis.

*Gummatous infiltration* of the rectum is a somewhat frequent, severe, and clinically important affection. It is much more common in women than in men, taking place in the "submucosa above the internal sphincter." It has frequently caused a fatal result in persons who failed to show post-mortem specific lesions in other viscera, and hence it is to be classed as one of the ravages. The result of the gummatous infiltration is the production of a funnel-shaped stenosis of the rectum which narrows from below upward. Above the stenosis, and directly dependent upon it, there is dilatation of the rectum and the descending colon. Here may also be found ulcers—some specific, and others the result of mechanical pressure exerted by the fecal accumulations.

**Symptoms.**—The clinical features are for the most part those of a gradually induced stenosis of the rectum. At first there may be hemorrhages, suggesting internal hemorrhoids. The action of the bowels is irregular, and is followed shortly by a tendency to dysenteric diarrhea, with pains, tenesmus, and scanty stools containing mucus and pus. Prolapse of the rectal mucosa may occur, and, owing to the presence of small hemorrhoids, the true nature of the case may be overlooked. The disease is most distressing, and leads slowly and gradually to extreme emaciation and asthenia. Death may be due to the latter or to some complication (perforative peritonitis, etc.).

**Diagnosis.**—This may be aided by a clear history of associated syphilitic symptoms or of specific lesions, including amyloid degeneration. A sure demonstration can only be made by rectal examination. The examining finger feels the sharp edge of the cicatricial ring. Cancer of the rectum can readily be eliminated on account of the absence in syphilis of the "crater-like" ulcer.

## SYPHILIS OF THE LUNGS.

While undoubted cases occur, syphilis of the lungs is rare indeed.

**Pathology.**—The cases are pathologically divisible into four forms: (a) Gummy tumors; (b) Interstitial pneumonia; (c) Brown induration; (d) Fetal pneumonia.

(a) **Gummy Tumors.**—These appear as yellowish-white scattered nodules, varying in size from a cherry-pit to a hen's egg. Their centers are dry and caseous-looking, and their peripheral zones fibrous. They are relatively thicker set near to the root of the lungs. Cicatricial bands may be seen connecting not only the separate nodules, but stretching outward to the thickened pleura. Such growths may undergo softening and ulceration, thus forming a cavity that rarely attains to large measurements; or, on the other hand, in favorable cases the fibroid changes and cicatrization may lead to recovery.

A primary lesion is atrophy of the alveolar walls, with hyaline degeneration of the capillaries (Councilman). Broncho-pneumonia (not distinctively syphilitic) may be associated.

(b) **Interstitial Pneumonia.**—This is a fibrous infiltration, showing a predilection for the right lung. Its chief seat is the root of the lung, whence it extends along the bronchi and vessels, and usually involves a part of one or more lobes. Occasionally its starting-point is the pleura, from which the process advances along lines corresponding to the inter-



lobular tissue. Bronchiectasis may be noticed. Gummata may also be associated, or may have been present and been practically obliterated during the process of cicatrization. I have seen an instance in which the merest vestige of gummatus material remained.

(c) **Brown induration**, simulating exactly that which is seen in association with organic valvular diseases, may be observed, but it may have no necessary connection with syphilis.

(d) **Fetal Pneumonia** (*Virchow's White Hepatization*).—This is peculiar to the new-born, in which miliary gummata first occur, followed by hepatization of large zones or an entire lung. The chief changes are an infiltration of the alveolar walls, while the air-cells are filled with desquamated epithelium; on section the tissue presents a grayish-white appearance.

**Symptoms.**—From what has just been stated it is clear that a certain limited number of cases present symptoms and signs that simulate ordinary ulcerative phthisis, but do not show bacilli in the sputum, and hence have no connection with genuine phthisis. There is another group of cases in which the picture presented to view is almost identical with that of fibroid induration though usually giving a distinctly syphilitic history. I am not prepared to say that there is an acute syphilitic broncho-pneumonia analogous to acute pneumonic phthisis, though I fail to see any reason why malignant syphilis may not attack the lung and take that form. The symptoms may be too few and too mild to afford ground for suspicion.

**Diagnosis.**—If a suspected case is treated early and accurately, the result may serve to corroborate the diagnosis, which is at first far from being final.

*Bronchiectasis*, dependent upon syphilitic peribronchitis or interstitial pneumonia, cannot be discriminated from other forms of that disease except there be a clear history of infection, and unless associated scars or active syphilitic lesions coexist. *Pulmonary tuberculosis* cannot be distinguished from *pulmonary syphilis* without a careful microscopic examination of the sputum. Moreover, it must not be forgotten thatluetics often develop ulcerative phthisis, and hence these affections are often combined.

#### SYPHILIS OF THE SPLEEN.

*Pathologically*, syphilis of the spleen is to be classed with the general adenopathy of the disease. According to the statistics of Sée (relating to hereditary syphilis) and of Avanzini and Schuchter (relating to acquired syphilis), in about 25 per cent. of the cases of secondary syphilis hypertrophy of the spleen may be noted. This augmentation begins from two to four weeks after the appearance of the chancre, and gradually increases, persisting throughout the secondary period; it is not, however, observed during the tertiary stage. It is often accompanied by localized pain—syphilitic pleurodynia (Besnier).

#### SYPHILIS OF THE CIRCULATORY SYSTEM.

**The Heart.**—The pathologic divisions are—(a) *Gummata*, which attack chiefly the walls of the left ventricle. They are usually encysted.

(b) *A Fibro-sclerotic Myocarditis*.—The process begins in the perivascular tissue and proceeds from the vessel walls outward (Mracek). It is diffuse, as a rule, and leads to narrowing of the lumina of the coronary arteries and their branches or to aneurysmal bulgings, but the pathologic effects of these lesions are seldom detected clinically. Sudden death may occur.

(c) *Syphilitic Endocarditis*.—The changes are of the fibro-sclerotic variety, and not of the more acute verrucose or warty type. The symptoms to which the lesion gives rise are depicted under Organic Valvular Disease.

### SYPHILIS OF THE ARTERIES.

Two forms are recognized: (a) *Obliterating Endarteritis*.—Here the syphilitic product consists chiefly of proliferated subendothelial tissue, which encroaches more and more upon the lumen of the vessel—a fact to which the disease owes its name. This so-called “Heubner’s degeneration” is not peculiar to syphilis, but, as Osler says, “if, however, there are gummata in other parts, or if there be gummatous periarteritis in adjacent vessels, the process may be regarded as syphilitic.”

(b) *Gummatous Periarteritis*.—This results in larger or smaller nodules or ovoid masses that may encircle the artery. Among common seats are the cerebral and coronary vessels, the growth starting in the adventitia and proceeding outward. According to the views of syphilographers of the present day, it is to be classed with the ravages of the disease, and hence is not due to the syphilitic virus.

Syphilis of the arteries has an important etiologic bearing upon atheroma and aneurysm (*vide* Diseases of the Arteries).

### SYPHILIS OF THE KIDNEYS.

Renal syphilis belongs chiefly to the tertiary stage, though it may appear in the secondary.

**Pathology.**—(a) *Amyloid degeneration* is a common renal lesion.

(b) *Granular atrophy* (Jaccoud).

(c) *Gumma*.

**Symptoms.**—Except in the case of amyloid degeneration the conditions are impossible of correct diagnosis. Wagner describes a special form which he calls *acute syphilitic glomerulo-nephritis*. Clinically, it is characterized chiefly by hematuria, and ends rapidly with uremia.

### SYPHILIS OF THE JOINTS.

The following division of the affection is made by Hutchinson of London:

(1) *Synovitis* appears during the secondary stage, but soon clears away under appropriate treatment, leaving no traces behind.

(2) *Perisynovial gummata*.

(3) *Arthritis*, due to osseous nodes or gummata in the neighborhood of the joints.

(4) *True Chronic Synovitis*.—This is the most common form of syphilitic arthritis.

(5) *Syphilitic chondro-arthritis* (Virchow).

The last four forms belong to the tertiary lesions.

**Symptoms.**—At the outset it is to be borne in mind that a joint-affection that does not yield to specific treatment is not necessarily non-syphilitic.

Perisynovial gumma attacks most frequently the tissues around the knee-joint; it is very chronic in its course and is more commonly seen in women than in men.

Arthritis due to osseous nodes has a special diagnostic feature in the severe nocturnal pains. The fourth form of syphilitic arthritis (true chronic) is the most common among the types due to acquired syphilis, while the symmetric synovitis of the knees occurring about puberty is perhaps peculiar to the congenital cases. The latter is apt to follow interstitial keratitis, and fortunately clears up rapidly under treatment.

### SYPHILIS OF THE TESTICLES.

The lesions are of two forms: (a) *Gummata*.—These produce hard, usually nodular, swellings, either single or multiple, and of moderate size, that occupy the substance of the testicle and sometimes the epididymis.

(b) *Interstitial Orchitis*.—This is a fibro-sclerotic change that leads to slow, gradual atrophy. Though bilateral, it is usually more marked on one side than the other.

**Diagnosis.**—In gummatus orchitis the swelling of the testicle is painless, usually nodular, and feels much like a scirrhus growth. Rarely it ulcerates, forming a fungous testicle. The frequency of syphilitic involvement of the organ forms a leading factor in the diagnosis.

In *tuberculous disease* the history and associated lesions differ from those of syphilitic orchitis, and the epididymis is generally affected. Atrophied testicles may be due to congenital syphilis. In such instances typical scars, eye-affections, and the characteristic physiognomy are usually to be noted. Hydrocele may owe its origin to the same cause. Atrophy of the testes may lead to impotency and sterility. Such instances are not to be mistaken for the results of metastasis in mumps.

**General Diagnosis of Syphilis.**—Perhaps sufficient has been said regarding the importance of obtaining a correct statement with reference to the primary infection. On failure to find evidence of a genital chancre, an examination for extragenital primary sores must be instituted, and the latter will be found to be by no means rare, even among children. The presence of a scar may betray the previous existence of a chancre in cases in which infection is denied or overlooked by the patient.

The secondaries are rarely puzzling, especially when the previous history is complete. There may be complicating eruptions. In Bulkley's records of 300 cases, 23 well-recognized affections of the skin were associated with syphilis. In this connection two facts need to be emphasized: first, that a syphilitic eruption, either macular or papular, may rarely cause troublesome itching; and second, that a patient with a syphilitic eruption may experience itching due to another cause—namely, eczema or scabies.



*Inherited syphilis* may be diagnosticated on the appearance in a child under five months of snuffles and the characteristic skin-eruptions. *Syphilis hereditaria tarda* may be recognized from a retrospective view or from the presence of active lesions and symptoms, or from both of these factors.

The recognition of the *tertiary manifestations* of acquired syphilis embraces these points: 1. The consideration of the fact that obscure cases in general and atypical symptom-groups are often due to the syphilitic taint. 2. Direct information or proof, as the result of careful inquiry, to show that the primary and secondary stages (either one or other, or both) have transpired. 3. The evidence presented by the patient and to be obtained by the careful objective examination of the eyes (for iritic adhesion, etc.), throat and skin (for scars), bones (for necrosis and nodes), and the testes. 4. Certain symptoms are significant, such as nocturnal pains, paralysis of the single cranial nerves, double deafness without otorrhea, etc. 5. The therapeutic test may aid in doubtful cases.

The presence of scars constitutes a most important factor in making a retrospective diagnosis. Recent scars are pigmented, and exhibit a slow, progressive clearing up, until, from four to eight years after infection, they are wholly decolorized. On the other hand, as pointed out by Hyde, eczemato-varicose scars remain stationary. These scars are apt to be found on the scalp and on the anterior surfaces of the legs. They may be single or multiple, and may exhibit certain defined shapes (semilunar, dumb-bell, etc.).

**General Differential Diagnosis.**—Numerous affections and conditions—local and general—are liable to be confounded with syphilis. Mere allusion to some of these common errors of diagnosis can be made here, while others must be omitted altogether:

(a) *The primary sore* of the lip has been mistaken repeatedly for cancer. The history and symptoms of syphilis, together with the therapeutic test, must clear up the doubt.

(b) Certain *skin-eruptions* (lichen, psoriasis, papular eczema, etc.) may be mistaken for the eruption of secondary syphilis. J. V. Shoemaker<sup>1</sup> details the differential diagnosis in a recent article, which the reader who desires full information may consult.

(c) Care must be exercised lest the *specific eruptive fevers*, especially the papular stage of small-pox, be mistaken for secondary syphilis.

(d) The syphilitic arthritis which may develop at the onset of the second stage must be discriminated from *rheumatic arthritis*—an easy task if only the attention be drawn to the primary lesion and the characteristic secondaries in cases of the former disease.

(e) Syphilis in the tertiary stage may simulate *chronic gout* or *rheumatism*, and unless there is definite evidence of syphilis on the one hand, or typical rheumatic symptoms and history on the other, the diagnosis may remain indefinitely uncertain. The therapeutic test may aid.

(f) *Periosteal nodes*, like those occurring in syphilis, may follow vaccination, small-pox, typhus and typhoid fevers. Here the history and associated phenomena furnish reliable data to effect a discrimination.

<sup>1</sup> *Medical Bulletin*, Nov., 1893.

(g) *Carcinoma of the tonsil* has often been diagnosed, and the tonsils have been excised when really the seat of a syphilitic lesion.

**Treatment.**—(a) **Prophylaxis.**—To prevent the transmission of hereditary syphilis infected persons should not marry within three years after the appearance of the primary sore. “Marriage should also be prevented when the patients have not been subjected to a thorough and prolonged treatment” (Porter). If at the end of the third year the patient presents a mucous patch, he must wait one year longer, and in the meanwhile be actively treated. A fresh outbreak of symptoms in a luetic patient demands immediate and active treatment.

Should a healthy mother bear a syphilitic child, she must not be allowed to suckle it. This precaution, though apparently contrary to the principle laid down in Colles’s law, is not superfluous, since the mother might be infected by any oozing fissures or condylomata upon the lips or in the mouth of the child if erosions of her nipple were to occur. Wet-nurses should not be employed for syphilitic children, but may be for non-syphilitic, even when the mother is affected. If syphilis appear in the mother during pregnancy, antiluetic treatment should be begun and persisted in even after apparent recovery. After the birth of the child, in such instances the course of treatment should be continued, if the child be nursed by the mother, with a view to medicating the milk.

As has already been stated, the most frequent mode of infection is irregular and illicit sexual congress, and it follows that absolute moral purity would go further toward the prevention of this widespread malady than any sanitary code or legal restrictions. Physicians cannot too strongly advocate continence. Should prostitution be regulated and controlled by the state? Experience has shown that but a slight control is exercised over the spread of syphilis in countries where systematic regulation of prostitution is attempted by the state. I am of opinion that the state should maintain some form of sanitary regulation and control, but, unfortunately, to render this efficient demands that prostitutes shall be officially registered. Such a sanitary supervision should consist in the examination of every prostitute at least twice a week, including a microscopic examination of the uterine and vaginal secretions, and the sending of every diseased prostitute to a hospital with a special department for such cases.<sup>1</sup> Palmer suggests that the female offender is usually not aware of the existence of a primary sore, while the male is; hence the latter should undergo inspection also. Inspection of prostitutes, however, unless rigid and careful, is absolutely valueless. Chancres are often concealed from view in the vagina or upon the lateral aspect of the os uteri. The maintenance of legal brothels, however, is not here recommended, either from a moral or hygienic standpoint.

(b) **Medicinal Treatment of Hereditary Syphilis.**—For syphilis of the new-born, mercury, either in the form of calomel (gr.  $\frac{1}{10}$ —0.0064, t. i. d.) or gray powder (gr.  $\frac{1}{2}$ —0.0324, t. i. d.), is to be employed. These babies must be hand-fed. The issue is almost unexceptionally bad.

When the first symptoms appear at the second or third month the above method of treatment is generally successful. Among the poorer classes no objection is made to mercurial inunctions, and these are often

<sup>1</sup> *The Berlin Commission on the Prevention of Syphilis*, Dec. 1, 1892.



preferable. The ointment may be rubbed into the arm-pits, thighs, or sides of the abdomen, which should be covered with a flannel roller. The hygienic details must be attended to, and especially must the parts be kept clean, and the mouth washed after nursing with a 3 per cent. solution of boric acid. *Syphilis hereditaria tarda* is best treated by the use of potassium or sodium iodid. To the iodid may be added mercuric chlorid in suitable doses, though the latter may, as pointed out by Roberts, often disagree. In addition to the specific therapy, tonic measures are usually indicated, and when employed are of the highest service.

(c) **Treatment of Acquired Syphilis.**—There is a specific plan of treatment which should be commenced as soon as the appearance of the secondaries has set the diagnosis of the given case at rest. This is the use of mercury, and rarely of potassium iodid also. The instances in which the latter alone is to be administered are among the rarest occurrences in medical practice. Fournier's "chronic intermittent treatment" of syphilis—which consists in continuous medication for two or three years with mercury and iodin alternately—is warmly advocated by some syphilographers, but the continuous mode is, in the opinion of most specialists, of greater advantage to the patient. Unless mercury disagree or the patient is exceedingly susceptible to its physiologic effects, I use it persistently during the secondaries, and later at intervals until the end of two years. It is a protracted course, and a protracted course only, of the specific treatment that suffices if we would obviate the dread ravages that otherwise are so apt to appear. I usually employ the protiodid (gr.  $\frac{1}{8}$ — $\frac{1}{5}$ —0.008–0.012, three times a day), and later the biniodid (gr.  $\frac{1}{30}$ — $\frac{1}{24}$ —0.0021–0.0027, three times a day). Hutchinson recommends the gray powder, given in pill form, combined with Dover's powder (*āā*. gr. j—0.0648), this pill to be taken from four to six times daily. I can speak from considerable personal experience as to the efficacy of this method. A well-known mixture, freely prescribed in many dispensaries, contains mercuric chlorid and potassium iodid in combination.

*Inunctions* of mercurial ointment (3ss–2.0, night and morning) produce excellent results, and it is advisable in cases in which the syphilids yield unsatisfactorily to internal dosage to suspend the latter at intervals of six or eight weeks and give a course of inunctions. Surely, they often hasten the disappearance of the more obstinate late secondaries, such as psoriasis (palmar), glossitis, etc. White advances the view that in the later stages, with the involvement of the deeper tissues, the combined use of inunctions over the affected region with potassium iodid internally often seems to have distinct advantages as compared with the administration of the "mixed treatment" by the mouth. It is necessary to omit the inunction once in seven or eight days for one day, and to take a warm bath to aid in the elimination of the mercury.

*The hypodermic* use of mercury in syphilis is to be adopted only when very prompt action of this agent is desired. Several preparations are used, and whether these are soluble or insoluble is a matter of little moment. The bichlorid takes first place, the dose being gr.  $\frac{1}{4}$  (0.0162), in 15 to 20 drops of water, twice a week. Calomel probably holds second place (dose, gr. j—0.0648, in 15 drops of glycerin, twice a week). Among other preparations employed are the peptonate of mercury and gray oil. All injections must be made deeply into the muscles. The subcutaneous



injection of sterilized serum from the blood of lambs and calves has been successfully practised by Tommasoli. The hypodermic medication in the mercurial treatment of syphilis is not unattended with dangers and accidents, though, fortunately, the latter are rare.

*The method of fumigation* has gained favor, particularly in the treatment of syphilis, in institutions on the Continent. Lane recommends that calomel (ziss—6.0) be put in a china bowl about half filled with water; a spirit lamp is placed under this, and the patient, "sitting above it wrapped in a cloak, has a deposit of mercury settle all over his body as the calomel is sublimed." He should remain wrapped in the cloak for one hour, take a fumigation once daily, and remain in-doors. From six weeks to three months are necessary to effect a cure.

If during the mercurial treatment the slightest evidence of salivation arise (tender gums, superficial glossitis, fetid breath, etc.), the administration of the drug must be interrupted for one week or ten days. The teeth should be cleaned thrice daily. *Hygiene* plays no mean rôle in the successful management of syphilis. The *diet* must be liberal, though green vegetables and fruits are not to be taken; and alcohol and tobacco are the two great enemies of the luetic.

*Auxiliary measures*, when the disease is associated with other lesions, are important. If syphilis occur in a tuberculous subject, it is of great value to add the potassium iodid to the mercurial, and, if active tuberculous lesions are present, cod-liver oil and creasote as well. Anemia and debility call for iron and a tonic plan of treatment generally. Attention should be given to the stomach, bowels, kidneys, and other internal organs, as well as to the nervous system.

*In women* the iodids should be suspended during menstruation if the flow of blood is excessive, but not the mercury. Says Mauriac: "During pregnancy specific treatment is well tolerated, and often requires to be pushed to a point a little short of intoxication for the good of both the mother and the child, close watch being kept upon the kidneys, suspending treatment at the first sign of albumin."

(d) *Treatment of Tertiary Syphilis*.—For most tertiary manifestations, including visceral syphilis, we have a therapeutic specific in potassium iodid. This should be used alone, the inunctions of mercury being added if the iodid fails to produce the desired result. I give the potassium iodid in a saturated solution, one minim being equal to  $\frac{3}{4}$  grain of the salt. I use gr. x (0.648) t. i. d. at the first dose, and increase the latter 1 grain (0.0648) each day until the manifestations for which it has been prescribed disappear or iodism is induced. It is best given in milk. In cases showing cerebral symptoms it is to be cautiously used, and it is then my custom to combine the iodid with potassium bromid.

*In hepatic syphilis* the mercurials are usually combined with iodids from the start, and particularly calomel if there be ascites or jaundice.

*In nervous syphilis*, especially in the graver forms, I begin with large doses (gr. xx—1.296, three times a day), and augment as above indicated. The limit of doses depends upon the effect produced. I have often found sodium iodid to agree better with the stomach than the potassium salt. Most syphilitics tolerate the iodids to a remarkable degree; on the other hand, a few show a marked idiosyncrasy to them. Among unpleasant effects are coryza, conjunctivitis with edema of the

eyelids, salivation, and certain skin-eruptions (erythema, urticaria, etc.). In this form of syphilis, as in the earlier stages, the specific treatment is made much more effective by attention to certain hygienic measures—fresh air, appropriate diet, bathing, exercise, and rest.

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## LEPROSY.

(*Lepra.*)

**Definition.**—A chronic, contagious disease, caused by the bacillus lepræ. It is distinguished by constitutional depression and, pathologically, by tuberculous masses in the muco-cutaneous surfaces, and by changes in the nerves.

**Historic Note.**—In 1889, Morrow stated that in India alone there were certainly not less than 150,000 lepers, while at present it is estimated that there are over 250,000. As in India, so in other regions, especially tropical, leprosy is on the increase, and its geographic distribution probably covers more than one-third of the entire surface of the globe. It is common in Africa, Brazil, in the East, and in Norway. In the Sandwich Islands the disease is of comparatively recent origin, and yet of great and increasing prevalence, a leper settlement having been established consisting of more than 11,000 cases. Leprosy is not unknown in America, and in Mexico it has existed ever since the time of Cortes (Morrow). Blanc states that there are 75 to 100 lepers in Louisiana alone. It was introduced into California and Oregon by the Chinese, and into Illinois, Iowa, Wisconsin, and Minnesota by Scandinavian immigrants. It has been imported from the Sandwich Islands to *Salt Lake City*, and from Normandy to Tracadie on the Gulf of the St. Lawrence, where the "disease is limited to two or three counties which are settled by French Canadians" (Osler). Sporadic cases have been met with in most of the larger American cities.

**Pathology.**—The bacilli grow and develop in clusters in the tuberculous nodules in the skin, residing within the epithelioid cells and leukocytes. These so-called lepra-cells are probably derived from the lymphatic vessels or capillaries, having been transformed by the bacilli. Surrounding the granulomatous masses is a layer of connective tissue. The bacilli are also found in the lymphatic glands, the spleen, and liver, but rarely in the blood. The nodular tumors form projections from the skin-surface, and, being poorly supplied with blood-vessels, they soon undergo caseation and absorption or are obliterated by dense connective tissue, which leads to the condition known as *facies leontina*. The pus-organism generally exercises an influence in causing suppuration with ulceration, which may manifest a marked destructive tendency. The changes in the internal organs or in the mucous membrane are identical with those above described.

*Nerve-lesions* are induced by the presence of the bacilli within and around the nerves. Here they first set up an irritation with hyperesthesia (neuritis), which leads to atrophy, with degenerative changes and the characteristic symptom, anesthesia.



**Etiology.—Bacteriology.**—In 1880, Hansen discovered the bacillus lepræ, which has since been proved to be the special agent of the disease. It strongly resembles the tubercle bacillus, but differential stains have been suggested by Unna and others. The British Leprosy Commission have shown that the bacillus can be cultivated, but inoculation experiments on animals have not as yet succeeded.

**Predisposing Causes.**—Every one is susceptible to leprosy, but the disease is most frequent between the twentieth and fortieth years, and is rare in childhood. Sex and latitude have little if any influence. Hereditary transmission probably influences about one-fortieth of the instances, according to the careful investigations of Zambaco. As pointed out by Bidentkap, leprosy is often rare in large cities, even though it is quite prevalent in the surrounding rural districts.

**Modes of Infection.**—The disease is doubtless transmitted by contact, but Widal and others, who have studied the disease as it exists in the Hawaiian Islands, think that leprosy is contagious only by inoculation. The effects of the accidental inoculation may either show themselves immediately or a long time may elapse before they are seen. Morrow's view, that, like syphilis, leprosy is generally transferred by sexual intercourse, receives abundant support. The possibility of transmission by vaccination must be also admitted.

**Clinical History.**—Two forms are recognized, the *tubercular* and the *anesthetic*, while a third or mixed type is described by some authors. Neither of the first two, however, runs its entire course without developing into a third or mixed form.

The incubation is usually long (three to five years—Hansen), but it may rarely be a comparatively short period. Vague prodromal symptoms are present for years, of which the chief are drowsiness, chilliness, irregularly recurring attacks of fever, debility, etc.

(1) **Tubercular Form.**—In the first stage there is a patchy, cutaneous erythema with a slight hyperæsthetic elevation of the affected areas. These are oftenest seen on the face or upon the extensor surfaces of the arms and knees. They may vanish after a while and leave the skin pigmented and anesthetic, and later the pigment may disappear, while white spots of corresponding size remain (*lepra alba*).

When the disease progresses less favorably tuberculous nodules (dusky-red or almost brown in color) develop in addition to anesthesia. The small ones soon disappear, while the large ones are either absorbed or break down and ulcerate—changes which, as they advance together with the slow healing process, produce marked deformities. The skin is greatly thickened and presents a scaly surface, and there is loss of substance in certain parts, while others are enormously enlarged (eyebrows, nostrils, lips, etc.). Among the many symptoms pointing to involvement of the mucous membrane are ozæna, hoarseness or even aphonia, and the signs of inhalation-pneumonia. To the last-named disease, as well as to ulcers extending deeply into the mucosa of the pharynx and larynx, death may often be ascribed. The end may thus be reached amid the evidences of extreme asthenia, if not as the result of gradual failure of strength and energy.

(2) **Anesthetic Form.**—In this variety the local symptoms point usually to implication of the nerves. At the onset there are pain and patchy



hyperesthesia, while minute bullæ, due to trophic changes, put in an appearance on the arms and legs. The muscles supplied by the branches of the affected nerve-trunk waste, and the superficial nerves feel thickened and nodular. Bright-red patches of vaso-motor congestion appear and soon become anesthetic, while the maculæ disappear. Anesthesia may proceed without the latter eruption. Dry, yellowish-white, scaly patches upon the trunk and extremities are also visible. Early their centers alone are anesthetic, but subsequently the loss of sensation spreads gradually until large areas, as well as healthy portions of the skin, are invaded, as in a case of Dehio's.

*Trophic alterations* reach an extreme degree. Bullæ of considerable size appear, and, bursting, leave perforating or destructive ulcers, usually upon the extremities. As the result of absorption, wasting, and necrosis great deformities are produced, such as contractures, exposure of the bones, ankylosis, etc. The hands often take on a claw-like form, and the fingers and toes may disappear (*lepra mutilans*).

**Diagnosis.**—The early diagnosis rests upon the presence of patchy erythema with hyperesthesia, followed by the development of anesthesia, with a disappearance of the macular eruption. Nodular neuritis is pathognomonic of anesthetic leprosy. In the advanced stages of either form confusion could scarcely arise. The nodular form of tubercular syphilis is distinguished by the distribution of the lesions, the history, and the frequent sensory nerve-lesions. Zambaco and others have claimed that *syringomyelia* and *Morvan's disease* are in most cases but forms of leprosy; but this has been disputed by Hoffman, Schlesinger, Sahli, and others. Syringomyelia depends on lesions of the central nervous system, while leprosy has its nervous lesions in the peripheral nerves; and on this basis the two may be differentiated. The first symptoms in syringomyelia are localized usually in the upper extremities. In leprosy they are generalized, and more often especially affect the lower extremities; also by the latter disease they are limited often to the area supplied by one nerve, and in the former to that supplied by one segment of the cord. In leprosy the tactile sense is usually lost, in syringomyelia usually not lost, etc. The individuality of *Morvan's disease* is disputed, and it is not impossible that many cases so diagnosed have been either leprosy or syringomyelia.

**Prognosis.**—Leprosy runs a very chronic course, lasting sometimes two, three, or more decades. The prognosis as to the final issue is hopeless, but the patient may live in comparative comfort for many years before the ravages of the disease cause great mutilation.

**Treatment.**—Here may be mentioned the fact that certain diseases are supposed to exercise a retarding effect on leprosy (pleurisy, pneumonia, variola, phthisis, etc.). Antagonistic inoculation, however, as practised by Beaven Rake and others, has been practically negative in its effects; and the same is true of the treatment by Koch's tuberculin, which has been tried by Arning, Babes, and others. No specific remedy or agent has been found for this disease. Phillippo, however, found that gurjun oil was serviceable, ulcers being rapidly cured by its application, with few exceptions: the oil may also be applied as an ointment to the swollen parts. Internally, chaulmoogra oil has been employed by Berge and Phillippo with excellent results, the dose being from 1 to

2 drams (4.0–8.0). It is sometimes administered in pearls (each containing  $\frac{1}{3}$  to  $\frac{1}{5}$  (0.199–0.333), in ascending doses, until the limit of tolerance is reached. The symptoms must be met as they arise, and the patient placed under the most favorable sanitary conditions. Surgical interference may become necessary in both varieties. Segregation of lepers has been instituted in certain localities with encouraging results.

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## GLANDERS.

(*Farcy.*)

**Definition.**—An infectious disease of equine origin, and caused by the bacillus mallei. It is rarely transmitted to man. Two leading forms are recognized—*true glanders*, which attacks the nasal cavities, and *farcy*, in which the chief lesions are cutaneous.

**Pathology.**—The characteristic lesions are new growths (granulomata, according to Virchow), which are usually nodular in character, though they may be diffuse. These masses soften and form ulcers when they occur on the nasal mucosa, and abscesses when they are situated subcutaneously. Microscopically, the nodular tumors are composed of cells—lymphoid and epithelioid—together with the specific bacillus.

**Etiology.**—The morbid changes above described are caused by a specific organism, the bacillus mallei, which resembles closely the tubercle bacillus, though it is a little thicker as well as shorter. It is non-motile. It can be readily grown, and as readily inoculated into horses, in which it produces the disease with every characteristic symptom. Perhaps the simplest method of staining the bacillus mallei “is to treat a cover-glass preparation with warm carbol-fuchsin, and then wash it off with a 2 per cent. solution of nitric acid.”

**Modes of Infection.**—The virus is, as a rule, transferred directly from the infected animal to man, hence the disease occurs almost invariably among persons who come in contact with horses (hostlers, coachmen, soldiers, horse-dealers, farmers, etc.). Transmission from man to man has been observed, but rarely. The medium of conveyance is either the pus or the nasal secretions, which may drop upon a wound in the skin or mucous membrane, however slight, and be absorbed. Males are infinitely more liable than females, owing to differences in occupation.

**Immunity.**—The disease is rare in man, most probably because of almost complete natural immunity. Singer has produced artificial immunity by intravenous injections of sterilized cultures of the glanders bacillus.

**Clinical History.**—The duration of the incubation period is from three to five days, and rarely longer. Both glanders and farcy may be *acute* or *chronic* in their course.

(1) **Acute Glanders.**—At first the signs of inflammation develop at the point of infection, lymphangitis and swelling of the adjacent lymphatic glands being associated. Fever and other evidences of general disturbance soon appear, and at the end of two or more days the nasal mucosa



becomes implicated, ulcers forming in the manner previously described, from which a fetid muco-purulent (sometimes blood-streaked) discharge takes place. Nose-bleed is a very common symptom. Usually a little later an eruption comes out on the face, the trunk, and the extremities, particularly about the joints. It is papular, quickly becoming pustular, and the pustules may dry up while fresh papules are developing—a characteristic feature. The face, particularly the nose, now swells, and a bluish-brown tumor covered with vesicles appears. Implication of adjacent mucous membranes—conjunctivæ, pharynx, mouth, etc.—is usual, and less frequently the bronchial and gastro-intestinal mucous membranes are involved. The ulcerative processes may extend until they touch the bones, setting up necrosis.

**Diagnosis.**—Cases have been mistaken for variola, but the history of exposure, mode of onset, the nasal symptoms, and the course of the eruption differ from those of the latter disease. In doubtful instances pure cultures should be made, and inoculated into the rabbit or guinea-pig. Death of the animal usually occurs within twenty-four hours.

(2) **Chronic Glanders.**—A rare disease which presents rather mild but vague general symptoms, such as muscular and arthritic pains, fever at intervals, asthenia, and progressive wasting, combined with the local features of nasal catarrh, with a muco-purulent discharge containing blood. Cough may be present. The *diagnosis* demands the making of pure cultures and of inoculation experiments.

(3) **Acute Farcy.**—In this form the virus is inoculated into the skin, which presents the chief symptoms, the nasal condition being in abeyance or absent. The primary lesion is of an aggravated type, accompanied by a large crop of cutaneous boils and abscesses, which often follow the lines of the lymphatics. Their favorite seat is in the vicinity of the joints. The constitutional symptoms simulate those of acute pyemia. The *diagnosis* is reached in the same manner as in preceding forms.

(4) **Chronic Farcy.**—Granulomatous tumors, resulting in abscesses, constitute the chief clinical peculiarity. The abscesses are situated primarily in the subcutaneous tissues, and generally in close proximity to the joints. As a rule, they open spontaneously and discharge indefinitely, first a thick, creamy pus, and later (if they do not heal) a thin fetid material. They sometimes form distinct ulcers, which extend in depth until the tendons and even the bones are involved.

**The general symptoms** simulate those of chronic glanders, the fever-curve being of the hectic type, particularly toward the close of the attack. About this time emaciation and prostration become extreme. The duration varies from ten to eighteen months, though death often results earlier from some associated disease or constitutional infection.

**The diagnosis** cannot be made without a clear history of contact with an animal known to be affected with the disease, or by the experimental method and artificial cultures. One of the products of the bacillus mallei is so-called “mallein,” which has been used by Nocard and others as a diagnostic agent in animals. Its injection into horses suffering from glanders is followed by a febrile reaction. Bonomé<sup>1</sup> does not consider mallein as a reliable diagnostic reagent. Schindelke injected more than six hundred horses with Forth's mallein, and his results show that

<sup>1</sup> *Riforma medica*, Naples, May 25, 1894.



a reaction of  $3.5^{\circ}$  F. ( $2^{\circ}$  C.) is an almost positive proof of glanders; a rise exceeding  $1.85^{\circ}$  F. ( $1.5^{\circ}$  C.) affords a strong presumption; while a rise of  $1.25^{\circ}$  F. ( $1^{\circ}$  C.) is suspicious.<sup>1</sup>

**Prognosis.**—Acute glanders and acute farcy are almost invariably fatal. The chronic forms, however, and particularly chronic farcy, end in recovery, under appropriate treatment, in nearly one-half the cases.

**Treatment.**—The primary lesion should be dealt with surgically, and thorough disinfection followed by cauterization is highly recommended. Bayard Holmes advocates the opening of fresh abscesses and the scraping out of old ones under an anesthetic. A supporting plan of treatment, by generous feeding and judicious stimulation, is to be adopted, and the symptoms are to be met as they appear. The product, "mallein," has been recommended as a specific in this disease, but even Bonomé, who reports an instance (occurring in a lad of sixteen and a half years) in which recovery followed its use, contends that its curative properties have not yet been perfectly demonstrated.

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## ACTINOMYCOSIS.

("Big-jaw," "Lumpy-jaw," etc.)

**Definition.**—An infectious disease of cattle, less frequently of man, caused by the ray-fungus (actinomyces), which grows in the tissues, developing a mass with secondary chronic inflammation and metastatic growth, as well as a pyemic condition, due to a mixed infection with pyogenic organisms.

**Historic Note.**—In 1877, Bollinger gave the first description of the ray-fungus, which he had observed in the disease known as "big-jaw" in the ox. It remained, however, for Israel of Berlin to discover the fungus in man one year later. In 1879, Ponfick showed clearly that actinomycosis in man and cattle was one and the same disease. Murphy, who described the first case of actinomycosis hominis in America, states that up to January 1, 1891, there had been reported 250 cases of the disease in man, while up to the present date more than 500 cases have been reported.

**Pathology.**—A macroscopic mass is produced, consisting of a central fungous mass from which threads of mycelia radiate in all directions, producing the ray form of growth. Individual growths are of the size of a millet-seed, but their aggregation may result in masses as large as an orange. They are generally yellowish and of tallowy consistence. Projections occur from the surface with club-shaped extremities.

Microscopically, the little or single ray-like tumors show straight or wavy branching filaments (*supra*). Their development is accompanied by the growth of dense adjacent connective tissue. In addition, abscesses containing yellow granules in the pus occur, but these are secondary. The usual lesions are not the same as those described as occurring in beasts. In man the lesions are those of chronic pyemia, and consist of a metastatic abscess-formation. This is probably due to mixed infection, pus-organisms being added to the actinomyces.

<sup>1</sup> *The American Year-book of Medicine and Surgery*, 1896, p. 1013.

**Bacteriology.**—The organism of the disease probably belongs to the cladothrix variety of fungus, and may be cultivated, though with difficulty. The finer threads may readily be stained with anilin colors. The club-shaped projections, however, do not take these stains, so that when examining sections Gram's method may be used. Rabbits and cows have been successfully inoculated with cultures of the actinomyces.

**Modes of Infection.**—Infection generally takes place through the mouth, teeth, and pharynx; and much less frequently the gate of entrance for the virus is the air-passages or the skin. It is generally introduced with the food or drink, and Bostroem, from a study of 32 cases, has come to the conclusion that the poison enters the economy by means of the injected grains of some cereal. It is to be recollected that infection cannot occur through a normal mucous membrane or skin, but that a wound, however insignificant, is essential.

**Clinical History.**—(1) **Oral Actinomycosis.**—The patient often complains of toothache, dysphagia, and of difficulty in opening the jaw. The latter symptom may be owing to induration of adjacent muscles, and is a very characteristic sign (Partsch). At the angle of the jaw a swelling appears which quickly passes into suppuration; later it opens (first externally, then into the mouth) and discharges pus containing little yellow masses. If not properly treated, extension of the process takes place in a downward direction, and thus in succession the lower jaw, the structures of the neck, the esophagus, clavicle, lungs, heart, and even the abdominal organs, may become involved.

The upper jaw may be the primary seat of infection, and if so the base of the skull may be perforated and the disease attack the meninges and brain. Bollinger has seen primary actinomycosis of the brain. In these instances the extension may take place in the direction of the spinal column, setting up caries.

(2) **Pulmonary Actinomycosis.**—I am satisfied that primary pulmonary actinomycosis is comparatively rare, and that oftener the lungs are invaded secondarily to actinomycotic disease elsewhere, especially in the oral cavity. The disease begins with pain in the side, and more often upon the left, due to pleurisy. There are cough and a peculiar (often fetid) expectoration, together with general wasting. A microscopic examination of the sputum, if made with care, reveals the actinomyces and furnishes a pathognomonic symptom.

In some instances the symptoms are identical with those of disseminated tuberculosis (Brigidi), though generally the disease is unilateral. There is irregular fever, due chiefly to suppuration.

The **physical signs** may be those of chronic bronchitis merely, but there are, in not a few cases, extensive destructive changes of variable character (abscess, broncho-pneumonia, etc.) which modify the signs accordingly. In primary pulmonary actinomycosis an extension to adjacent organs, and also metastatic growths and abscesses in various parts of the body, are the rule.

(3) **Intestinal Actinomycosis.**—The condition may be primary or secondary. The organism grows upon the mucosa of the intestine and excites a proliferation of the underlying connective-tissue cells, and the formation of submucous nodules. The latter ulcerate, and perforation

of the serous coat of the bowel may occur, inducing peritonitis. Pericecal abscesses have been formed in like manner.

The **symptoms** point to intestinal catarrh, there being some gastric disturbance, with irregular and recurring attacks of diarrhea. The actinomyces has been detected in the stools. Secondary metastatic growths (rarely) and abscesses may arise in other organs (liver, spleen, ovaries, etc.), but it is to be recollected that the primary seat of infection may also be the spleen, liver, or other viscerae.

(4) **Cutaneous actinomycosis** rarely occurs. The skin presents chronic suppurating ulcers which show the presence of the ray-fungus, and the condition bears a close resemblance to a lupus patch, as in a case reported by Darier and Gautier.

**Diagnosis.**—This rests solely upon the finding of the actinomyces. The wooden hardness of the tissues beyond the borders of the ulcers or sinuses, the hardness of the neighboring muscles in oral actinomycosis, and the yellow granules in the pus are all significant, but merely corroborative. To detect the actinomyces, says Warren, sections may be stained in Ziehl's carbol-fuchsin from fifteen minutes to half an hour, and then decolorized in a 1 per cent. picric-acid solution until the whole section has a yellow appearance. Dehydrate and mount. The fungus appears as a brilliant red aster, while the surrounding tissues are yellow.

**Course and Prognosis.**—The course is chronic. Mild cases may recover in from six to nine months or earlier, the oral form being perhaps the most favorable. Schlange, after an analysis of 60 cases of actinomycosis, concludes that pulmonary actinomycosis may terminate in recovery, though rarely. Cases in which a pyemic condition develops generally reach a fatal termination within a comparatively brief period of time. Death usually results from amyloid degeneration and wasting.

**Treatment.**—This is mainly surgical. The removal of the parts involved and disinfection with acid-sublimate solution are the best measures. Köttwitz records marked success from incision of the abscesses followed by cauterization with solid silver nitrate. Billroth in a case of abdominal actinomycosis communicating with the bladder effected a cure by the use of fifteen tuberculin injections. Internally, the potassium-iodid treatment as first recommended by Thomassen in 1885 has been attended with success in the hands of many observers. The treatment is most efficacious when decided iodism is produced.

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## ANTHRAX.

(*Malignant Pustule; Splenic Fever; Wool-sorter's Disease, etc.*)

**Definition.**—An acute, infectious disease, caused by a special bacillus and clinically accompanied by the development of a characteristic pustule (boil) and blood-poisoning (*external anthrax*). The disease likewise affects the gastro-intestinal tract and the lungs (*internal anthrax*). Both forms are derived chiefly from herbivorous animals, it being especially prevalent among sheep and cattle. The existence of anthrax in



the United States, in Asia, Russia, and parts of Europe has been denied, but it occurs rarely, and Bard has described its ravages in California.

**Pathology.**—Post-mortem rigidity is marked. The blood is dark and thick and coagulates poorly, and in it, particularly in the spleen, as well as in the liver, kidney, and lungs, one may find the spores in great numbers.

Besides the local lesions of the skin (*i. e.* ulceration, gangrene, edematous infiltration), and besides the degeneration of the heart, kidneys, and liver that is common to the severe and rapid infectious diseases, the especially striking lesion is the constant and great enlargement of the spleen. This may occasionally attain four times the normal size, and rarely it has ruptured. It is always greatly distended with blood.

In cases in which the intestines are affected the bowel shows hemorrhagic infiltration and gangrene, and the mesenteric and retroperitoneal glands are enlarged and hemorrhagic.

**Etiology.**—**Bacteriology.**—The special agent is the bacillus anthracis or its toxin, or both. Gratia and Jonne give as the microscopic characteristics of anthrax, as seen in the blood, the following: (1) The anthrax bacillus has the form of a rod of a length varying from  $5\mu$  to  $20\mu$ , and in breadth from  $1\mu$  to  $1.5\mu$ . It is broken up into short articulations from  $1.5\mu$  to  $2\mu$  long, placed end to end like the sections of a tenia, the ends of each articulation being slightly swollen, giving the appearance of a bamboo cane; (2) clear spaces, appearing like a biconcave lens, exist between the ends of the articulations, and result from the slight concavity of these ends; (3) a capsule, often distinctly marked, surrounds the rod, seeming to form a protoplasmic support for the individual articulations. These threads of anthrax bacilli stain best with Löffler's blue. They grow readily on various media (agar, gelatin, potatoes, etc.) into interlacing thread-like filaments which distinctly show spore-formation, the threads assuming the appearance of strings of beads. Toxic substances are developed in the culture-media. Remarkable instances are recorded of the prolonged vitality of anthrax spores in dry or moist earth and in drinking-, sea-, and sewer-water. They resist desiccation, many of the germicides, and boiling water even for a few minutes, and facts such as these explain why anthrax is stamped out with such difficulty in a locality where once the spores have developed. The latter are not produced within the body either of the inferior animals or man. Inoculations are followed by the production of the pustule of anthrax.

The *virus* (spores) gains entrance into the human body through the skin (slight wounds, abrasions, or scratches), the intestines (with food), or through the lungs (rarely). The sting of insects (mosquitoes, flies) may also transfer the poison to man. Among disposing factors, however, occupation is most influential: persons who come into direct contact with affected animals (hostlers, butchers, shepherds), and workers in factories who handle the hair or hides of such animals, being especially exposed.

**Immunity.**—Pasteur's well-known protective inoculation with attenuated virus has been extensively practised in anthrax localities with very favorable results. Hankin, by means of albumose, which he separated from cultures, claimed to have produced immunity against the most

virulent anthrax. Peterman, however, reinvestigated the question of immunity by the albumose of anthrax, and found it to be without protective action, except in the case of cultures on ox-serum, which, when injected in large quantities into the veins, conferred immunity, though only temporarily.

**Clinical History.**—The period of incubation is from one to three days. Two leading clinical types are distinguished:

(1) **External Anthrax.**—(a) *Malignant Pustule.*—At the point of infection (the hand, arm, neck, or face, or other exposed part) a small papule first appears, and develops into a vesicle of considerable size with bloody contents. This vesicle breaks, leaving a characteristic dark-bluish or black scab (anthrax), and encircling the primary vesicle an areola of miliary vesicles may be noticed. The base of the original vesicle now becomes swollen and indurated, and this brawny edema spreads rapidly to the adjacent tissues until an extensive area is involved. The neighboring lymph-glands may or may not be inflamed; if so, they are apt to be connected with the pustule by red lines corresponding to the lymph-vessels and veins.

Severe general disturbances accompany the local disorder in the course of a couple of days, and comprise fever, decided prostration, sweats, splenic enlargement, and delirium tending toward coma. If recovery occur, the edematous swelling subsides and the black scab is cast off, sometimes leaving a granulating surface. On the other hand, in unfavorable instances collapse develops, and the case ends fatally between the fourth and eighth days. In such instances intestinal symptoms (diarrhea) or nervous phenomena of aggravated type may attend.

(b) *Anthrax Edema.*—In a certain proportion of the cases the systemic infection is out of proportion to the local disturbance, the latter consisting of an edematous swelling without the presence of an eschar. The eyelids, lips, tongue, and upper extremities may be the seat of extensive swelling, though there is no change in the color of the skin. This is a dangerous condition, and sometimes results in extensive gangrene.

(2) **Internal Anthrax.**—(a) *Intestinal Mycosis.*—In this form certain general, indefinite symptoms are the primary features, such as headache, pains in the limbs, anorexia, languor. Soon acute gastro-intestinal symptoms supervene, sometimes preceded by a chill. As a rule, vomiting occurs, followed by abdominal pains and diarrhea, and the stools often become bloody. Hemorrhage may also occur from other outlets. Other symptoms, as dyspnea, marked cyanosis, and restlessness, are noted, followed sometimes by stupor, general convulsions, or spasms of single muscles or groups of muscles. There is only moderate fever, but the spleen is enlarged, and the pupils may be dilated. Death is preceded by collapse.

Interesting epidemic outbreaks of internal anthrax have occurred, due both to drinking-water derived from infected wells and also to diseased meat. Murisier has related the history of an epidemic in which 200 persons fell ill after eating meat from a certain cow. The animal was quartered by a butcher who had previously slaughtered an ox afflicted with anthrax, and had not disinfected his instruments; four days after this 25 persons were attacked by the disease.



(b) *Wool-sorter's Disease*.—This occurs among the operatives in factories in which imported wool or hair, mostly from Russia and South America, is sorted, and to produce the typical affection the infection must be swallowed or inhaled in the form of dust. Mixed cases, or those showing both external and internal anthrax, may be met with among workers in curled-hair establishments and the like. The onset is sudden, with a chill that is accompanied by pains in the back and legs, prostration, and a sharp rise of temperature to 102° or 103° F. (39.4° C.). The *local* symptoms may either be chiefly pulmonary or gastro-intestinal. The former consist in dyspnea, chest-pains or feelings of constriction, cough, and rarely the physical signs of bronchitis; the latter comprise vomiting and a diarrhea that is followed either quickly or after some days by collapse, with marked lividity. Nervous symptoms, delirium, convulsions, or coma are often prominent, and particularly when death is imminent; but a fatal ending may suddenly take place while the mind is unclouded. The usual course ranges from one to five days.

(c) *Rag-picker's Disease* ("Haderkrankheit").—This has been identified by Eppinger as the same form of disease as "wool-sorter's anthrax." It occurs among the rag-sorters in the paper-mills near Graz. Infection occurs in the respiratory tract. The symptoms observed are high fever, followed by collapse, with depression of the body-heat, painful and paroxysmal cough, cyanosis, very weak heart, together with the signs of pleuritic effusion and consolidation of the lung. Systemic infection may ensue. In 8 cases the bacillus of anthrax was isolated.

**Diagnosis.**—The history (occupation, etc.) and the appearance of the malignant pustule in external anthrax leave little room for doubt. The diagnosis, however, should be confirmed by an examination of the contents of the pustule for the presence of bacilli, and if found they should be cultivated and inoculated upon the guinea-pig or rabbit.

The recognition of internal anthrax is more difficult, but the condition may be suspected if the more characteristic pulmonary or gastro-intestinal symptoms, together with those of systemic intoxication, develop in persons whose occupation entails exposure to infection. In these doubtful cases the only safe course is to show the presence of the bacilli in the blood, and, unfortunately, this is generally impossible till death is near at hand.

**Prognosis.**—In external anthrax occurring in healthy persons the disease often pursues a favorable course; moreover, radical surgical measures have decreased the death-rate decidedly. Internal anthrax, however, is a deadly affection. As regards "wool-sorter's disease," Bell, who first recognized the affection, states that those who survive for one week usually recover. Herbivorous animals are more susceptible to anthrax than is man, and in them the mortality-rate is relatively higher—from 70 to 80 per cent.

**Treatment.**—In malignant pustule it has been recommended to destroy the point of infection by caustic or by the hot iron, and then dust with powdered mercuric chlorid with a view to destroying the mass. It seems to me, however, that, unless the pustules be small and remain so, removal by excision should be preferred, and Klein and others have reported recoveries after removal of the primary focus of infection. In preventing extension of the brawny edema hypodermic



injections, several times daily, of a solution of carbolic acid at points a little distance from the site of the pustule have given the best results. Hallopeau recommends that in order to prevent extension the neighboring structures be bathed with a 10 per cent. solution of carbolic acid (first dissolved in alcohol) in oil or glycerin. He applies the same to the surface of the anthrax. Internally, stimulants, antiseptics, and nourishing food constitute our chief reliance.

In internal anthrax efforts at treatment avail nothing. Osler wisely recommends active purgatives with a view to removing the infecting material, but further than this the internal treatment differs little from that employed in the external form.

## HYDROPHOBIA.

(*Rabies.*)

**Definition.**—A specific, infectious disease peculiar to carnivora and to a less extent to herbivora, which may be communicated to man by direct inoculation. It is characterized by slight fever, spasm of the larynx and pharynx, delirium, a short stage of paralysis, coma, and, in the great majority of cases, by death.

**Pathology.**—The facies, pharynx, and esophagus may be congested, the latter organ being sometimes markedly edematous; pulmonary congestion has also been noticed. The mucous membranes may show here and there points of hemorrhage, and Fitz has observed blood-extravasations into the perivascular spaces of the brain. Soft thrombi may fill the cerebral vessels, especially the veins, while the blood has a dark color and its clots lack firmness.

The chief lesions are found in the medulla, and particularly in the region of the respiratory center, in which the nuclei of the hypoglossal, pneumogastric, and glosso-pharyngeal nerves are seated. On microscopic examination Gowers found the cord in rabies to show merely hyperemia in the gray substance;<sup>1</sup> in the medulla, however, he observed in addition a cell-infiltration of the perivascular lymph-sheaths and sometimes of the adjacent tissues as well. Small areas infiltrated with leukocytes (miliary abscesses) were noted, and scattered through the adjacent tissues were seen small round-cells in unusual numbers. Fitz and Shattuck found as the most constant change an infiltration of the adventitia of the veins with small round-cells. Rarely, the kidneys may show cloudy swelling. The cadaver putrefies rather early.

**Etiology.**—No micro-organism is known to be the special agent of the disease, but that it is of microbic origin cannot reasonably be doubted. The virus is contained chiefly in the saliva, and has been successfully inoculated into other animals both by means of this secretion and of the blood of affected animals. Pasteur has found the poison abundantly present in the nerve-centers, and has transferred the disease by taking bits of brain-substance or medulla derived from an infected animal and inoculating them into healthy subjects.

<sup>1</sup> Golgi has described important changes observed in the cord structures, but these have not as yet been confirmed by other observers.

The usual mode of infection in man is through the bite of a rabid animal, and in an immense majority of cases (about 90 per cent.) the dog is the offending party. The cat, wolf, cow, and horse also suffer from the disease, though less frequently than the dog, and in rare instances only do they communicate the disease to man. The skunk is also liable, and its bite has often transmitted rabies, especially to persons sleeping in the open air or in tents which the animal can enter. The virus gains access to the system through the broken skin, and not through the mucous membranes.

*Susceptibility to the poison* exists in about one-half the instances in which persons are bitten by rabid animals, though in some cases this apparent immunity may be owing to slight or even non-infection.

**Clinical History.**—The *incubation-period* lasts from six weeks to three or four months, though in young subjects and in cases in which the infection is severe the symptoms develop earlier. Certain prodromal symptoms are manifested as a rule, and generally last only a day or two; I have, however, seen two instances in which melancholia, due probably to the dread of what might follow, showed itself immediately after the reception of the bite and persisted. The usual premonitory symptoms are headache, loss of appetite, sleeplessness, great depression of spirits, and sometimes darting pains that radiate from the seat of the bite. The adjacent lymph-glands may become swollen, and slight difficulty in swallowing is experienced.

Following the *invasion* are two stages: (1) **The Stage of Excitement.**—The patient wears an expression of the most intense anxiety. Hyperesthesia is present and attains to a marked degree, and the special senses exhibit the keenest vigilance, a noise or a draft of air often causing great psychic disturbance or a violent reflex spasmodic contraction of the larynx. Quite early the mere sight of water is dreaded by the patient, and forms a characteristic feature of the disease. This symptom has given the name hydrophobia to the disease, and springs from the fear of inducing a painful spasm of the larynx. The patient has thirst which he cannot assuage. There may be maniacal excitement, and the spasmodic contractions of the larynx may become so strong as to excite urgent dyspnea, with the emission of curious sounds. The muscles of the mouth may also exhibit convulsive movements, causing the patient to make snapping sounds; these, however, are secondary. There is associated great restlessness, with frequent lateral rolling of the head, and foaming saliva may be ejected from the mouth. The symptoms occur in paroxysms, and during the intervals the patient is generally free from excitement. There is fever as a rule, the temperature ranging from 100° to 102° F. (37.7°–38.8° C.) or over, but it may be absent; the pulse is moderately accelerated and is sometimes irregular, and toward the end of this stage the reflex spasms of the respiratory apparatus develop spontaneously. Mental aberrations may set in, and melancholia often leads to suicidal tendencies.

(2) **The Paralytic Stage.**—In the concluding stage the patient passes from a condition of debility into actual unconsciousness or coma, without spasms. This lasts from twelve to eighteen hours, during which cardiac asthenia rapidly increases, and life soon ends by syncope.

In man there is a *paralytic form* of rabies, but it is rare as compared



with the delirious or psychic type. Thirty cases have been reported by Gamaleia, and, according to this observer, the paralytic type is apt to follow deep and multiple bites. The paralysis begins near the part bitten, and spreads until it becomes general, finally involving the respiratory centers. In rodents quiet madness ("dumb rabies"), without maniacal excitement, is the rule.

The **diagnosis** is readily made, owing to the fact that the history of recent infection is usually obtainable. The hyperesthesia, the fear of water, the reflex spasms on attempting to swallow, accompanied by dyspnea and great mental agitation, form a very characteristic group of symptoms. *Hysteria* may be misleading, since convulsive movements may follow attempts at swallowing, but here the previous history suffices to explain the true nature of the case.

The name *lyssophobia* has been given to cases that simulate, but have no relation to, hydrophobia, and Mills has advanced the warning that, with however so suggestive symptoms following a dog-bite, the given case cannot be assumed to be a case of hydrophobia until other possibilities are excluded. It is highly probable that there is a form of hydrophobia which is the result of the wide publicity given to genuine and suspected cases alike. The characteristic symptoms may be present, but they are comparatively mild and the affection does not develop. This so-called *pseudo-hydrophobia* appears only in neurotic and hysterical subjects, and runs a longer course than does the disease itself. Recovery is the rule, and yet I feel convinced that even a lyssophobic condition growing out of fear of the disease may cause death. Burr reports an interesting case of the kind that occurred in Osler's clinic, attended, however, with recovery.

**Prognosis.**—Few if any cases of rabies in man recover if the disease be allowed to develop. Preventive measures (efficient and early cauterization, and more especially the Pasteurian antirabic inoculation) have been the means of reducing the frequency of the disease.

**Treatment.**—**Prophylaxis.**—Upon the reception of a bite thorough disinfection, followed by cauterization of the wound with caustic potash, etc., is a measure that can be quickly carried out. The wound is then to be kept open for a period of four or five weeks, and steps taken to carry forward prophylactic inoculation with precision.

*Preventive inoculation* as perfected by Pasteur is a precautionary measure of the utmost importance. This famous investigator, after discovering that the virus of the disease could be obtained in a pure state from the central nervous system, showed that its virulence undergoes modification by passage through animals. Thus the potency of the virus is increased by its inoculation from rabbit to rabbit (by placing bits of spinal marrow beneath the dura mater), the period of incubation at the same time growing shorter, till at last it is but seven days. On the other hand, the virulence is decreased or attenuated as the result of similar experiments upon the monkey. Pasteur also found that if fragments of the spinal cord were suspended in a dry atmosphere they lost gradually their virulence and finally became inert. From these an emulsion is prepared which is employed in the antirabic inoculations in man. In this way he secured a virus of known and reliable strength, and with this he could readily render the dog refractory by inoculating



with very weak virus; then, by increasing from day to day the virulency of the inoculations, complete immunity was established.

*Protective inoculation* in man was first employed by Pasteur in 1885, and is carried out by injecting the emulsion hypodermically. "The patients are first inoculated with a cord fourteen days old, and the inoculation is repeated daily for nine days, each time with a cord one day fresher. In winter the oldest cords used are five days old, and in summer cords that have been drying for four days are also employed. The preceding is the ordinary treatment" (Warren).

For patients who have been bitten on the face, hands, or bare feet, as well as for those who have been bitten long before commencing treatment, the special preventive method, the so-called "intensive treatment," is applicable. Briefly, this consists in eliminating some of the inoculations of intermediary strengths, thus lessening the number of injections, and also in administering the latter at shorter intervals than in the usual method of treatment. The success of the Pasteur method is almost universally attested, and his own claims, that few persons properly inoculated subsequently suffer from rabies, are generally conceded.

*The established affection* defies all known methods of treatment. Our aim should be to diminish the intensity of the painful spasms and the psychic disturbances. The patient should be isolated from sounds, light, and excitement of every sort. Food, as a rule, must consist of nutrient enemata, though by the local application of cocain the sensitiveness of the throat may be diminished sufficiently to enable the patient to take liquid nourishment (Osler). For controlling the spasms chloroform by inhalation is most effective; chloral internally and morphin hypodermically may also be tried with advantage. The patient's anxiety is best relieved by a cheerful demeanor on the part of the attendants.

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## TETANUS.

(*Trismus; Lockjaw.*)

**Definition.**—An acute, infectious disease caused by the tetanus bacillus. It is characterized by painful spasms, affecting first and chiefly the muscles of the jaw and neck (*trismus*), and secondly those of the trunk, especially the extensors of the spine and limbs (*opisthotonos*). The disease may be idiopathic, though more often it is traumatic. In certain institutions and certain localities (*e. g.* eastern end of Long Island) it occurs endemically, and among new-born children it may prevail epidemically (*trismus neonatorum*). To this latter form the colored race in hot countries (West India Islands, etc.) is especially liable.

**Pathology.**—No constant post-mortem lesions have been found. The majority of authorities contend that the virus acts chiefly upon the nervous centers of the medulla and the cord, where obvious traces of inflammation (and sometimes of softening of the gray substance of the cord) have been noted. According to Brown-Séquard, the characteristic lesions are consequent upon an *ascending neuritis* starting from the

wound, and it is true that the nerves often present traumatic lesions with redness and swelling of the neurilemma. Tetanus neonatorum often shows inflammation of the umbilicus.

**Etiology.—Bacteriology.**—In 1885, Nicolaier discovered the bacillus of tetanus, and in 1886, Rosenbach first found it in man. It is a long, slender rod, at one end of which appears a swelling due to the formation of a spore in that locality, thus giving the organism an appearance like that of a pin or drumstick. The bacilli are easily stained by Abbott's method, and are purely anaërobic. Pure cultures can be made, but with difficulty, since other varieties of bacteria are found in association, and if pure cultures are injected into animals, typical tetanus follows. Brieger has obtained the poison from sterilized cultures of the bacillus in the pure state, and termed it "tetanin"—a most virulent poison in the minutest quantity. The bacilli are most probably limited to the point of infection, and here develop the toxin (a toxalbumin).

**Modes of Infection.**—In the outer world tetanus bacilli are found to be both numerous and widely distributed. They abound in the earth (garden-soil in particular), putrefying liquids, manure, in rubbish and dust of streets and houses, etc. The fact that the bacillus of tetanus is anaërobic (cannot grow in the presence of oxygen) explains why tetanus in man is a comparatively rare disease, notwithstanding the widespread dissemination of the parasite, and also why it is most apt to follow punctured and contused wounds. It may, though less commonly, follow wounds of any sort. It may be assumed that in the immense majority of the cases an injury, however slight, serves as a gate of entrance for the poison. The *locality* of the injury is almost always on the extremities, particularly on the hands and the feet; the disease is most common *in warm climates*; and as regards *age* it is most common between ten and thirty, if we except tetanus neonatorum. Idiopathic tetanus may follow exposure to cold or sleeping on the damp earth, and in tetanus neonatorum the infection may be communicated either by careless nurses or by dirty dressings of the stump, etc. (Papienske).

**Immunity.**—Behring and Kitasato have rendered animals immune by the injection of cultures of the bacillus after the addition of iodine trichlorid to diminish their strength, and this serum has been successfully used to protect others against tetanus.

**Clinical History.**—The duration of incubation depends upon whether the given case pursues an *acute* or a *chronic* course. In acute tetanus it lasts from one to two weeks, while in chronic the first symptoms usually appear after the second week. In idiopathic tetanus the symptoms generally appear shortly after exposure to the special causes.

**Symptoms of Acute Tetanus.**—(1) Mild prodromal symptoms (languor, headache, etc.) may precede the more intense characteristic phenomena, which develop gradually. At first the patient complains of stiffness and tension in the muscles of mastication and back of the neck, and soon tonic spasm of the masseters renders the facial muscles more or less immobile and locks the jaws (*trismus* or *lockjaw*). The rigidity of the cervical muscles is shown by the retraction of, and by attempts at raising, the head. The physiognomy is distinctive; it is immobile, the forehead being often wrinkled and the corners of the mouth retracted,



producing a peculiar smile (*sardonic grin*). Next there is an involvement of the muscles of the body, first inducing rigidity of the trunk (orthotonos), and then the spine is bent or bowed and the convexity presents anteriorly (opisthotonos). Lateral arching of the body also occurs, though rarely (pleurosthotonos). The belly-muscles are hard and board-like, and their contractions may throw the body forward (emprosthotonos). The arms generally remain movable, but the legs may be rigidly extended. The position of the body is one of constant rigidity, but from time to time convulsive seizures of variable duration occur, causing most agonizing suffering, thoracic oppression, dyspnea, and more or less cyanosis, due to interference with the respiratory function (especially spasm of the glottis). Sharp lancinating pains occur at the base of the chest and point to contraction of the diaphragm. "Convulsive dysphagia" (as in hydrophobia) is rarely observed. These paroxysms may be spontaneous, though more often the spasms are reflex, due to the action of external irritants (generally slight in character). The reflexes are increased. The intellect remains clear. Profuse perspiration is a significant symptom.

Fever of a moderate degree is generally present. The temperature, however, may suddenly leap to  $110^{\circ}$  or  $112^{\circ}$  F. ( $43.3^{\circ}$ – $44.4^{\circ}$  C.), forming an ominous symptom, these extreme elevations of temperature being probably due to paralysis of the centers that regulate bodily heat. Conversely, fever may be absent throughout the attack, and a post-mortem rise of temperature be seen which lasts for a short period. The pulse is generally quickened, and in the worst cases may become very rapid (140 to 160 beats per minute), small, and irregular. The urine may be suppressed or its passage impeded by the muscular contractions. The bowels are constipated.

(2) *Chronic Tetanus*.—The same symptoms are manifested as are seen in the acute form, but the condition does not progress so rapidly. In some instances the symptoms soon become aggravated, to be followed, however, by periods of decided relief from the painful spasms, so that during the latter the patient's strength can be maintained by means of stimulants, etc. Intervals of partial freedom from the excruciating pains grow longer in favorable cases, until finally the period of convalescence may be reached. Relapses, however, are common.

(3) *Cephalic tetanus* (first described by Rose) usually follows injuries to the head and particularly to the face. Its most characteristic symptoms are rigidity of the masseter muscles, spasm of the pharyngeal muscles, causing dysphagia, chronic contraction of the muscles of the neck and abdomen (rare), and paralysis of the facial nerve on the same side as the injury. The latter symptom is due to local infection by a toxin. In this form, particularly if the course be chronic, recovery sometimes takes place, occurring in about 25 per cent. of the instances, according to Willard's statistics.

**Diagnosis.**—In view of the usual history, the predominating feature—trismus—together with the early appearance of rigidity at the back of the neck, will, as a rule, render the diagnosis a simple one.

*Strychnin-poisoning* is distinguishable, but often great care is needed in making the distinction. The following points belonging to each may be contrasted:



## TETANUS.

## STRYCHNIN-POISONING.

*History.*

Reception of a wound, generally followed by a period of incubation.

Ingestion of strychnin, followed immediately by the symptoms.

*Mode of Development.*

Begins with lockjaw ; later spreads downward (the arms and hands escaping).

Begins with gastric disturbance or a tetanic contraction of all the extremities.

*Symptoms.*

Reflex spasms not present at the outset.

Violent convulsions present from the onset.

Rigidity is persistent, except in the chronic form.

Intervals of complete relaxation occur.

The course is prolonged into days or weeks.

Course is brief, terminating in death or recovery.

Cultures made from the discharges of the wound show the bacillus tetani.

Examination of the gastric contents shows strychnin.

*Tetany* gives rise to a spasm of long duration affecting the extremities (hands in particular) and the larynx, with absolute intermissions; it is also characterized by a peculiar posture.

*Hydrophobia* is discriminated from tetanus by the history of a bite from an animal, by the predominance of the reflex spasm of the respiratory apparatus, by the intensity of the psychic disturbance, and by the absence of lockjaw and opisthotonos.

**Course and Prognosis.**—In the acute form the course is brief, rarely exceeding ten days, and the prognosis is most unfavorable. Death results from asthenia, heart-failure, or asphyxia (during the paroxysm). According to Richter's statistics, 88 per cent. of military cases are fatal. In idiopathic or rheumatic cases the mortality-rate is under 50 per cent. Chronic tetanus gives a less grave prognosis than does acute. In the new-born recovery is so rare that when it occurs the diagnosis may be called into question.

**Treatment.**—In traumatic cases the wound must be disinfected and thoroughly cauterized. In order to do this effectively, the agents employed must be brought in contact with every portion of the wound, so that punctured wounds must first be laid open. Excision of the wound, and even amputation, may be advisable in some cases. The fact that the deadly poison is developed at the site of infection gives to the local measures supreme importance in the treatment of tetanus.

The patient should occupy a secluded room with little light and a carefully regulated temperature. A single nurse will suffice, and all sources of external irritation should be avoided. A nourishing diet is demanded, and rectal feeding must be instituted as soon as it is found that food cannot be administered *per oram*, or the food may be introduced by means of a small stomach-tube or catheter passed through the nostril. Stimulants should not be spared when the heart's action becomes quick and feeble. In one of my own cases hypodermic injections of strychnin and digitalis probably saved the patient's life. The spasms are best controlled by chloroform-inhalations, and during the intervals the patient should be kept under the influence of morphin, administered

subcutaneously. Among other remedies that have sometimes been successfully employed are chloral hydrate and Calabar bean. The former may be exhibited in rectal injection (gr. xl—2.59 at a dose), to be repeated at intervals of six to eight hours until the spasm is overcome. The heart, however, must be carefully guarded. Rarely, good results have been obtained from the use of potassium bromid, curare, nitrite of amyl, belladonna, and cannabis indica.

*Tetanus-antitoxin* has been recommended for the cure of the disease, and is prepared both in fluid (antitoxin serum) and dry form. The former deteriorates in quality, in consequence of which fact Roux and Vaillard, as well as Tizzoni and Cantani, have adopted the method of drying the serum. A dried preparation is also obtainable from Merck and his agents in the form of tubes containing from 4 to 5 grams each; at the time used it may be dissolved in water or in glycerin. Of Tizzoni's antitoxin 2.25 grams are to be given at the first dose, and 0.6 grams at subsequent doses. Huebner<sup>1</sup> has studied carefully the immunizing value of Tizzoni's tetanus-antitoxin, and finds that in the strength recommended by its author it is powerless to cure cases of tetanus in the human being if grave or if brought under treatment at a late period. Kanthack's and Kneass's<sup>2</sup> recent statistical analysis also shows that the question as to its value is still *sub judice*.

## INFECTIOUS DISEASES OF UNKNOWN ETIOLOGY.

### MUSCULAR RHEUMATISM.

(*Myalgia*.)

**Definition.**—A common, painful disease of the muscles and of the structures to which they are attached (fasciæ and periosteum), probably due to an attenuated form of the virus of acute articular rheumatism. Leube contends—and very properly, I think—that muscular rheumatism is a general disease with local symptoms. The latter may be seated in different parts of the body, and in this way give rise to a number of leading sub-varieties, and it may either accompany acute and chronic rheumatism or it may be experienced as an independent disease. I have also met with several instances in which it followed joint-rheumatism, and Leube has seen it precede the latter. This observer noted on one occasion that half the beds in his ward were occupied by patients with this disease, giving evidence of an epidemicity. Certain authors, however, believe that the affection is a neuralgia of the sensory nerves of the muscles.

**Pathology.**—In fatal cases (these are exceedingly rare) the affected muscles show a swelling of the fibers and more or less granular change. In long-standing cases an atrophy of the muscles, due to trophic disturbance, may be observed.

**Etiology.**—Among the disposing influences that are most important in the causation of the affection are—(1) The *rheumatic diathesis*

<sup>1</sup> *Deutsch. med. Woch.*, Aug. 16, 1894.      <sup>2</sup> *Journ. Amer. Med. Assoc.*, July 18, 1896.

(appropriate soil); (2) *Heredity*; (3) *Exposure* to cold, damp, and strong air-currents, especially after heavy exercise or during free perspiration; (4) *Sex*, owing to the more frequent exposure of men while following their occupations; (5) *Age*. It is met with at all ages, but acute and subacute forms most frequently occur among children and young adults, while the chronic form most frequently affects elderly persons; (6) *Previous attacks* increase the susceptibility to the disease, as in acute articular rheumatism.

**Symptoms.**—In the majority of instances the clinical symptoms are local. Out of 200 cases Leube found fever in about one-third, the temperature rarely exceeding 102° F. (38.8° C.) for two days in duration, and far more often remaining normal. In one-sixth of Leube's cases there was a cardiac murmur, that disappeared under treatment in one-half of this number. The most conspicuous local symptom is the pain, which is sometimes sharp, lancinating, and paroxysmal, though it may be deeply seated, dull, and constant. The changes are essentially those of myositis. In the acute form there is often an extensive round-cell infiltration of the connective tissue, with swelling and partial degeneration of the muscular fibers and the formation in them of vacuoles. In the chronic form there is a proliferation of the interfascicular connective tissue, and the muscle-fibers exhibit slight granulation and increase in the number of nuclei. The pathology of this condition is very indefinite. It is aggravated at night in most cases, and also by contraction of the affected muscles, by weather changes, and in acute forms by pressure. In long-continued cases pressure with the broad side of the hand usually affords relief. The duration of the affection is exceedingly variable; it usually ranges from a few hours to several days, but may rarely pursue an apparently endless course.

**Leading Clinical Varieties.**—(1) **Lumbago** (*Myalgia Lumbalis*).—This is the most common form, and may be taken as the type of the myalgias. The onset is sudden, sometimes intensely so, and the lumbar muscles are exceedingly painful and sensitive. Motion, such as stooping or turning the body or rising from the sitting position, causes intense exacerbations of pain. The affection occurs most frequently in laboring men, its course being brief, as a rule, and recurrences frequent.

(2) **Pleurodynia**.—This term implies involvement of the intercostal muscles, and less frequently of the pectorals and the serratus magnus. It is unilateral, and oftener affects the left than the right side, and causes untold suffering, since it is constantly aggravated by the normal respiratory excursions. The pain is also intensified by pressure, reaching, etc., and by movement of the trunk, sneezing, and coughing. Fortunately, it is not a very frequent affection. Similar symptoms may be occasioned by traumatism in which the fibers of the thoracic muscles are lacerated, and there is also great danger of confounding pleurodynia with costal periostitis and with pleurisy.

(3) **Torticollis** (*Myalgia Cervicalis*).—Here the muscles, some or all, on one side of the neck, and at times the throat, are implicated. The head is held toward the affected side, so as to relax the group of muscles involved, and on attempting to turn it the patient rotates his entire body in a pivot-like manner. The complaint is frequent in young persons.



(4) **Cephalodynia.**—By this term is meant rheumatism of the head-muscles of the scalp and fasciæ. It is by no means an infrequent condition, and may be either general or local, being sometimes limited to the frontal, temporal, or occipital muscles. The pain is severe and greatly increased on motion of the scalp.

(5) Other terms descriptive of localized forms of muscular rheumatism are employed: (a) *Omodynia* (myalgia of the deltoid); (b) *Dorsodynia* (involvement of the muscles of the upper part of the back, etc.); (c) *Abdominal rheumatism* (myalgia of the muscles of the abdomen); (d) *Rheumatic myositis* of the extremities.

**Diagnosis.**—This is assured by the etiologic influences and the presence of pain, which is greatly increased by muscular contraction. The presence of fever does not exclude the affection. It differs from *neuralgia* in that there are no painful points, and in that firm pressure with the broad hand often affords relief. *Dermato-myositis* must not be confounded with muscular rheumatism. Unverricht first distinguished the former from the latter, showing that there are present pain and swelling of the muscles, as in muscular rheumatism, but additionally redness (erythema) and hyperesthesia of the skin, while the joints usually escape. Rovere, however, reports a case occurring in the course of diabetes mellitus in which there was a joint-inflammation resembling that of rheumatism. Of general symptoms, the chief are fever and physical prostration. The spleen is enlarged, and angina and hemorrhages have been noted. The disease is obviously infectious, probably septic in nature, and occurs in fatal as well as in the mild or favorable forms. *Dermato-myositis*, unlike muscular rheumatism, which is more common among men, is seen more frequently in women, especially servants.

The **prognosis** is good, the disease never directly endangering life, though a person may be more or less incapacitated for work by muscular rheumatism.

**Treatment.**—Severe and acute forms demand the use of opiates internally and anodyne and hot applications externally. When cases are seen early, morphin, administered hypodermically, may serve to relieve the pain and cut short the disease. In acute cases the salicylates and other antirheumatic remedies are to be employed. Hot fomentations give comfort, and the Turkish bath may end the attack if it can be used sufficiently early. The hot-water bag, sponging with water as hot as can be borne, or dry heat in the form of bags filled with heated salt or heated hops, will all do good service. For the dull pain which is so distressing in some cases of torticollis the affected muscles may be covered with flannel, over which a warmed flatiron may be passed for a few minutes. This is an efficient expedient. For lumbago acupuncture is highly commended. Needles of from three to four inches (7.5–10 cm.) in length (ordinary bonnet-needles, sterilized, will do) are thrust into the lumbar muscles at the seat of the pain and withdrawn after five or ten minutes (Osler). Blisters have been recommended, but I have tried them frequently without beneficial effects in any case. In subacute and obstinate cases I have recently obtained good results from the use of a 20 per cent. ointment of salicylic acid freely rubbed into the skin. Active friction with anodyne and stimulating liniments (the latter when pain is

not great) is worthy of trial. Massage and electricity (the constant current in particular) are sometimes efficient, and in chronic cases potassium iodid, guaiacum, and arsenic (the latter in small doses) should be tried. The same measures of prophylaxis are to be adopted as in chronic rheumatism, and the condition of the general health must also be looked to, every endeavor being made to maintain the proper quality of blood and perfect nutrition.

#### CHRONIC ARTICULAR RHEUMATISM.

**Definition.**—An affection of the articular structures which develops slowly and gradually and may be dependent upon the same causes as the preceding forms. Rarely it is a sequence of acute or subacute attacks.

**Etiology.**—(a) *Age* predisposes to the affection. Though it may appear at any age, the greatest number of cases is furnished by the years from forty to sixty. (b) *Sex* exerts a slight influence, the disease being observed most frequently among females. (c) *External agencies*, as poverty and occupations which entail exposure to cold and dampness, act as predisposing influences. (d) *Heredity* may operate to favor its development.

**Pathology.**—The joints, as a rule, do not show pronounced gross lesions, there being some degree of synovial injection and also some, though not much, effusion. Inflammatory thickening of the articular and periarticular structures (capsule, ligaments, sheaths of the tendons, etc.) with contraction, is noted, and is a change which deforms and stiffens some joints to a certain extent. Superficial erosions of the cartilages may also be witnessed, and, as stated under the Clinical History of Acute Articular Rheumatism, muscular atrophy supervenes in long-standing cases of arthritis. The probable causes of these important changes have been pointed out in connection with the latter disease. When the shoulder-joint is the seat of chronic inflammation, this muscular atrophy (affecting chiefly the deltoid) reaches its highest degree of development.

**Symptoms.**—The involved joints may not present any markedly visible evidences of disease, and perhaps the most prominent local symptom is pain, increased often at night as well as by approaching cold or damp weather. Both the larger and smaller joints are involved, though the former to a greater degree, and yet, though usually multiple, the disease may be limited to one joint (knee, hip, shoulder, etc.). The joints are somewhat swollen, at times slightly reddened, tender upon pressure, and their mobility is generally restricted. Pain and stiffness are most marked in the morning hours (after rest), and often largely disappear with each returning evening (after use). All the local symptoms are subject to exacerbations and remissions. A peculiar crepitation may be elicited on applying the hand over the affected joints during motion, and eventually ankylosis, with some degree (usually slight) of distortion of the joints, may occur.

The general features are usually conspicuous by their absence. No fever is present, and, in most instances, there is no serious impairment of the general health. On the other hand, as the result of constant suffering, a wretched general condition with marked anemia and debility



may finally be reached, such patients often passing sleepless nights and suffering severely from dyspepsia. Chronic endocarditis may develop along with the chronic articular changes—a not uncommon association, though frequently the history of a previous attack of acute rheumatism is also obtainable, to which the endocarditis may be attributed (for the differential diagnosis of this disease *vide* Arthritis Deformans).

**Prognosis.**—Full recovery is, with but few exceptions, out of the question. A cure may rarely be effected if the case come under appropriate treatment in the incipient stage. The disease, however, rarely shortens the duration of life, though it may do so by interfering with the nutritive processes, the latter effect resulting from loss of sleep (due to pain) and inability to take active exercise.

**Treatment.**—(a) **The local measures** hold first place. The affected joints should be enveloped in flannel at all times, and underneath the latter may be applied cold cloths, and the whole covered with oiled silk. On the other hand, sponging the joints frequently with hot water also furnishes good results, relieving decidedly the pain and stiffness. Blisters have been employed, but I have failed to see any benefit from their use, except in the cases in which effusions were present. In removing the latter they are most efficacious. In the absence of synovial effusion the thermo-cautery is to be preferred to blisters, and for the swelling and stiffness massage with passive movement affords excellent results. Massage is also valuable when atrophy of the adjacent muscles exists; and in these so-called “rheumatic paralyses” electricity is an important help. The application to the joints of iodine and stimulating liniments is more or less serviceable.

(b) **Hygienic Measures.**—The diet should be nutritious and ready of digestion, since dietetic errors, with their usual baneful consequences, tend to aggravate the arthritic condition. The patient should adopt and continue in moderately active exercise until compelled to omit it on account of the advancing joint-lesions. Cold spongings of the skin-surface, followed by active friction, has a good effect in that it lessens cutaneous sensitiveness.

(c) **Internal remedies** do not control the morbid process directly, although arsenic, iodine, potassium iodide, guaiacol, and other agents are much used for this purpose, but their effects are usually limited, and never brilliant. It should be our aim to maintain the general health at a maximum level by the employment not only of the sanitary means before alluded to, but also by tonics (iron, quinine, strychnine, etc.). I have found a course of cod-liver oil, continued for a long period of time, the most serviceable form of internal medication.

(d) In general terms **hydrotherapy** is an important adjuvant to the treatment.

The thermal springs whose waters are alkaline or contain sulphur, and of which the hot springs of Arkansas and Virginia, and the Richfield Springs, New York, furnish good examples, have been strongly advocated, and sometimes prove curative in their effects. I have seen excellent results from the methodic use of hot-water baths at a constant temperature (100° to 105° F.—37.7° to 40.5° C.), combined with passive motion and careful manipulation of the affected parts. If the latter be adopted, every precaution must be used to avoid exposure to cold or



draft during and after the baths, which should not be prolonged beyond ten minutes.

### WEIL'S DISEASE.

(*Acute Febrile Jaundice; Fiedler's Disease.*)

**Definition.**—An acute febrile disease, probably specific in origin, and characterized by jaundice, remittent fever, and muscular pains. It usually runs a definite course and terminates by lysis.

**Pathology.**—During the comparatively recent studies of the post-mortem lesions occurring in this disease very little has been noted. The liver and spleen are sometimes the seat of an active hyperemia, and occasionally some gastro-intestinal irritation is present. The cortical substance of the kidneys is swollen and mottled, and the epithelium of the tubules and glomeruli shows cloudy swelling.

**Etiology.**—The exciting cause of the disease is probably some specific microbic agent, but as yet no bacillus has been finally shown to be responsible for the affection. Jaeger claims that it is due to infection by the *bacillus proteus fluorescens*.

**Predisposing Causes.**—Among these may be mentioned the following:

(a) *Age.*—The age of the patient usually varies from twenty to forty years.

(b) *Occupation.*—This seems to have little connection with the cause, though in a certain few cases the disease has been noted as occurring among butchers.

(c) *Sex and Season.*—Most of the recorded cases occurred in males and during the summer months.

**Symptoms.**—The disease is usually ushered in by a chill, followed by fever, headache, and pains in the muscles that may be agonizing. Jaundice usually appears on the second day, and may either be slight or very intense; if it be due to obstruction, the stools are gray-colored, showing the absence of bile. The fever is of the remittent type, running from ten to fourteen days and terminating by lysis. Nausea, vomiting, and diarrhea may also occur, but are rare. The liver and spleen are often enlarged, the latter being tender on pressure. The urine is febrile, high-colored, and often shows the presence of albumin, with tube-casts, and sometimes blood. In grave (but rare) cases cerebral symptoms, such as delirium, convulsions, and coma, may occur and prove fatal.

**Prognosis.**—The fatal cases on record are very few, and the prognosis, both as to life and recovery, is good.

The **treatment** is purely symptomatic.

### SCHLAMMFIEBER.

(*Oderflecken; Erndtefieber.*)

An epidemic disease that occurred in the basin of the Oder River and its branches near Breslau during the summer of 1891, supposedly in consequence of the floods of March. It was carefully studied by Müller of Marburg.

Nothing is definitely known concerning its pathology or its etiology, save the fact that it prevailed mainly among young persons who worked

in the recently flooded districts. It was not communicated by contact. Susceptibility to the disease was general, and seemed to be influenced neither by the sanitary surroundings, food, nor by the water. The first cases occurred in June, and by October the intensity of the epidemic had passed. The incubation was from eight to twelve days.

*Clinically*, the disease has not been satisfactorily classified, but Müller shows the resemblance in certain respects to Weil's disease, which may occur at times without jaundice. He would include both in the same group of diseases, and hence it is deemed unnecessary to describe the symptoms of "Oderflecken" separately. Few deaths occurred. No special form of treatment has been adopted.

### MALTA FEVER.

(*Mediterranean Fever; Rock Fever.*)

**Definition.**—An acute, infectious disease, caused most probably by the *Micrococcus melitensis*, and characterized by periods of remittent fever that are separated by shorter periods of apyrexia. It is endemic in Malta, and from time to time is encountered there, as well as at Naples and other Mediterranean ports, in epidemic form. No essential pathologic lesions have been identified with the disease. Hughes<sup>1</sup> noted an enlargement of the spleen and of the mesenteric glands, also irregular patches of congestion in the alimentary tract, and grave cases exhibited bronchitis or broncho-pneumonia.

**Etiology.**—The *Micrococcus melitensis* has been found in the organs twenty-one times, and is readily recognized morphologically and by culture. Bruce in two cases, and Hughes in four, reproduced the disease in monkeys by the inoculation of pure cultures of the organism. Young persons are most frequently affected.

**Symptoms.**—The incubation-period lasts from five to ten days, and the disease develops gradually like typhoid fever, though it is a distinct affection. Headache, anorexia, languor, and fever (often preceded by slight shiverings) are present, the fever being of the remittent type, though irregular, and lasting one, two, or three weeks. It then disappears, to be followed, after an apyrexial period of two or three days, by a relapse, with rigors, high fever, and delirium, and sometimes by diarrhea and increased prostration. The relapse frequently lasts from five to six weeks, and then usually gives place to convalescence. At the end of another week or two a second relapse somewhat similar to the first sets in, with rigors, an intermittent type of fever-curve, extreme prostration, and general rheumatoid symptoms. The latter may be so marked as to prohibit voluntary muscular movements of any kind. This most distressing condition may either terminate in recovery or, after the lapse of one or even two months, there may be a repetition of the whole group of symptoms.

In *grave cases* the temperature is continuous and death may occur in hyperpyrexia (Hughes). The symptoms and course of the disease resemble those of malarial fever, but the plasmodium is absent. The spleen is enlarged during the first five or six weeks, and then returns to its normal dimensions.

The **duration** is variable, obstinate cases lasting six months, but the mean length of stay in the hospital is from seventy to ninety days. There is marked anemia in protracted cases. The *mortality* is about 2 per cent.

The **treatment** is to be directed toward sustaining the strength of the patient by nourishing liquids and stimulants. The fever is unaffected by quinin or arsenic, and is to be met by hydrotherapy. Tonics, including iron, are needed to overcome the sequential anemia and debility.

### FEBRICULA.

(*Simple Continued Fever ; Ephemeral Fever.*)

**Definition.**—A brief febrile attack, unattended with definite local lesions, and of varied, often indeterminate, etiology. A true ephemeral fever is one that lasts about twenty-four hours, while the term simple continued fever or febricula is given to cases lasting a longer time—from three to six or more days.

The cases are so diversified with reference to their etiology and clinical relations as to make it desirable to group them roughly under several heads:

(a) A large group of cases in which a *gastro-intestinal disturbance* is the only assignable cause. The latter may take the form of indigestion due to cold or more often to errors in diet (particularly the use of tainted food-stuffs), accompanied by absorption of toxic substances; or it may consist of the gastro-intestinal catarrh so frequently met with in young children.

(b) Undeveloped or abortive forms of the *infectious diseases* (typhoid, typhus, rheumatism). These affections, particularly during times of epidemic prevalence, may run a brief course without manifesting any of their distinctive characters. In abortive types (particularly of typhoid fever) the invasion-symptoms are apt to be well marked, but the more characteristic features fail to appear. Thus diseases that ordinarily manifest a characteristic eruption (*e. g.* scarlet fever, measles, erysipelas) may run their course without doing so, or the eruption may escape observation.

(c) It may follow *exposure* to the summer sun or *excessive heat*, or *exhaustion of the nervous system*.

(d) It is not infrequently the result of a slight and unnoticed *localized inflammation* (tonsillitis, bronchitis, lymphadenitis, etc.).

(e) The *inhalation of sewer-gas or other noxious vapors* (such as emanations from decomposing organic matter) may produce an aberrant form of the fever, and sewer-air has been mentioned elsewhere as a cause of a mild form of sapremia (*vide* Septicemia).

**Symptoms.**—It is to be remembered at the outset that a single symptom, peculiar to all cases, is the fever. The onset is generally sudden, and especially in ephemeral fever, but it may be gradual; if sudden there is rarely either a chill or vomiting, while in neurotic children a convulsion may occur. The temperature ascends quickly to 102°–103° F. (39.4° C.) or over, pursues the continued type, and at the end of one, two, or more days subsides abruptly by crisis. There are accompanying symptoms, many of which are due to the fever, such



as headache, hebetude, mild delirium, flushed countenance, a full rapid pulse, anorexia, constipation, scanty high-colored urine, and, not rarely, herpes labialis. Defervescence may be attended with critical sweats, diarrhea, or a copious flow of urine. Special types (*e. g.* cerebral, gastric) may be observed, due to the predominance of the symptoms presented by individual organs or systems.

In another class of cases the access of simple fever may be less sudden, the maximum level attained being somewhat lower and the attending phenomena less acute and pronounced. The course is more protracted, though rarely exceeding a week or ten days, and the defervescence is not so abrupt.

The **diagnosis** necessitates the exclusion of other acute fevers. The affections from which it is most difficult to distinguish febricula are typhoid fever, scarlet fever, tonsillitis, larval pneumonia, and meningitis (in children). In febricula, however, there is an absence of local manifestations and of physical signs pointing to consolidation of the lungs; characteristic skin-eruptions are also absent.

The **prognosis** is good.

**Treatment.**—Few cases require treatment other than rest in bed and liquid nourishment for several days. Cooling drafts internally, and mild forms of hydrotherapy (spongings, ice-caps) externally, are indicated. If traceable to gastro-intestinal disturbance, a laxative usually proves beneficial and effective. It should be followed by intestinal antiseptics. Unless it is clear that the given case is non-infectious and non-contagious, isolation of the patient should be ensured.

## MILK-SICKNESS.

**Definition.**—A peculiar infectious disease, occurring both in man and in the lower animals, when it is known as “trembles.” The disease is unknown east of the Alleghany Mountains, but throughout many of the Western and South-western States it formerly prevailed very extensively, with fatal effect. It has, however, been almost exterminated as the result of denudation of the forests and the advancing cultivation of the virgin soil. It still prevails in parts of North Carolina (Osler), and until very recent times has been seen in certain parts of Illinois.

No peculiar pathologic lesions have been described.

**Etiology.**—It is believed to be due to a special poison derived from the earth, but as yet we are ignorant of its exact nature. Phillips claims to have found a spirillum in the blood.

**Modes of Infection.**—The disease attacks cattle most frequently (especially unweaned calves), horses, sheep, goats, and less often many undomesticated animals; wherever trembles prevails among cattle, milk-sickness is met with in man. It is thought that the poison is communicated to man in the milk, butter, and cheese, or in the flesh of infected animals.

Among disposing factors are the *seasons*, the disease being most frequent in the late summer and autumn. It is most common in adult life.

**Symptoms.**—The period of incubation may be short or long in duration, and prodromata, such as headache, anorexia, languor, and oncoming fatigue, may be noted. These symptoms increase in severity, and are soon eclipsed by the more characteristic features—nausea and

vomiting, a hot pain in the stomach, and a peculiar fetor of the breath. There is an unquenchable thirst, a swollen, tremulous tongue, and absolute constipation. Fever is present, but it is slight, and the surface-temperature is often below the normal. The nervous symptoms include restlessness, merging into mental dulness with marked indifference, and the latter condition passing in grave cases into a stupor that may deepen into actual coma. Convulsions may arise or the patient may drop into a fatal typhoid state.

The **diagnosis** rests chiefly upon the history (particularly upon the coexistence of "trembles" in cattle) and the exclusion of other acute intoxications.

The **prognosis** is generally favorable, though a fatal termination due to asthenia may occur within a few days of the time of the onset.

**Treatment.**—*Prophylaxis* consists in the avoidance of those foods that act as bearers of the disease. Apart from the use of supporting measures (appropriate diet and stimulants), we can attend only to the symptomatic indications. Medicated enemata should not be omitted.

### MILIARY FEVER.

(*Sweating Sickness.*)

**Definition.**—An infectious disease, characterized by copious sweats and a vesicular (miliary) eruption. In certain countries it has prevailed epidemically (France, England, Italy, Germany), and in 1887 a severe epidemic occurred in France. Schaffer<sup>1</sup> reports the occurrence of a recent epidemic in an Austrian province in the spring of 1893, lasting for nearly three months. Out of 5079 persons (the total population of the district), 159 suffered, as follows: 17 men, 14 women, and 128 children. At the present day it seems to be met with only in Picardy, in a few other French provinces, and throughout a limited area in Italy.

Neither have definite *pathologic lesions* nor the *specific exciting cause* been found. Among *predisposing influences* the following have been noted: (a) Most epidemics occur in spring and summer; (b) It is more common among women than men, and most frequent during the middle period of life. A large percentage of the entire population of an invaded district (usually limited in area) is attacked.

The **symptoms** that characterize miliary fever are fever with its usual accompaniments, irritation of the skin, a sense of oppression in the epigastrium, copious and persistent sweating, followed, on the third or fourth day of the disease, by an eruption of miliary vesicles.

The vesicles burst, and within forty-eight hours scaly desquamation is generally completed. In severe types the nervous phenomena (delirium, etc.) are grave in character; hemorrhages may occur, and at times fatal collapse may follow. Relapses are not uncommon.

The **prognosis** is affected largely by the character of the epidemic, the average death-rate being 8 or 9 per cent.

Quinin has met with almost universal favor as a remedy, but the expectant plan of **treatment** is the most appropriate, the symptoms being treated as they arise.

<sup>1</sup> *Wiener med. Blätter*, 1893, No. 32.

## FOOT-AND-MOUTH DISEASE.

(*Epidemic Stomatitis; Aphthous Fever.*)

**Definition.**—An acute infection of certain lower animals (cattle, sheep, pigs, goats, etc.), caused by a micro-organism as yet undiscovered. It is characterized by fever, by the appearance of vesicles and ulcers in the mucosa of the mouth, in the furrows about the feet and on the udder, and by the rapid development of asthenia and marked emaciation. Though a disease of mild character, its territorial range is so vast as to entail untold loss to European countries. Young animals or sucklings perish in great numbers on account of the deteriorated quality of the milk, which assumes a yellowish-white appearance and has a bitter, nauseating taste.

During epidemics of foot-and-mouth disease the poison may be transferred to man, in whom the disease is known as *epidemic stomatitis*, the poison generally being transferred by means of the milk. Boiling the latter destroys the virus, but rarely the infection may be transmitted through butter and cheese made from the milk of infected cattle. Communication by inoculation (while milking) may also occur. Whether the poison may be introduced into the human body by eating the meat of diseased animals is doubtful.

**Symptoms.**—The *incubation-period* lasts from three to five days. A rigor may mark the onset or merely slight shiverings, followed by fever and malaise, and soon vesicles, such as are described under Aphthous Stomatitis, appear upon the tongue and inner surface of the lips. The mouth is hot, the mucosa reddened and swollen, and salivation is present. A form of miliary eruption that may become pustular may also appear on the skin-surface, and particularly on the fingers and hands. Hemorrhages have been observed in severe epidemics.

The **diagnosis** is made with ease if the disease be prevailing at the same time among lower animals. The peculiar coincidence of the eruption in the mouth and extremities, sparing the rest of the body, has not been noticed in any other eruptive disease (Whittaker).

**Course and Prognosis.**—The course is mild and ends in about one week, the disease being very rarely fatal.

**Treatment.**—*Prophylaxis* requires the use of milk from healthy animals (cows or goats), together with measures looking to the care of the stables and isolation of diseased cattle. For *treatment* the reader is referred to the article on Aphthous Stomatitis.



## PART II.

# CONSTITUTIONAL DISEASES.

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### DIABETES.

(*Diabetes Mellitus.*)

**Definition.**—A nutritional affection, attended by an abnormal amount of sugar in the blood, and characterized clinically by persistent glycosuria, by polyuria, and by a progressive loss of flesh and strength.

**Nature of the Affection.**—This is still undetermined. Post-mortem lesions of different organs and structures of the body have been met with in diabetes—a fact that has given rise to a variety of views as to its nature, of which the following are the chief:

(1) That it is dependent upon *organic disease of the pancreas*, especially granular atrophy, or upon marked functional disturbance of this organ. It has been shown experimentally that extirpation of the pancreas is followed by diabetes, and yet, according to Minkowski and Lépine, if a small portion remains glycosuria does not result. On the other hand, Sandmeyer extirpated the pancreas of two dogs, leaving from one-ninth to one-fifth of the organ. The animals became diabetic—one four and the other thirteen months after the operation—and the first dog succumbed two months and the other eight months later.

It may safely be assumed that total loss of function always, and partial loss sometimes, leads to diabetes. Again, from the observations of Hansemann in the Berlin Pathological Institute and the Augusta Hospital, it would seem certain that the coincidence of pancreatic disease and diabetes occurs oftener than either diabetes or pancreatic disease alone, and, in truth, oftener than both these separate affections combined. Lépine and Martz have been able to produce a glycolitic ferment by treating the pancreas after their own special method, which need not be detailed here. This ferment is identical with that which is contained in the blood, and in the presence of which glycogen is assimilated; pancreatic diabetes occurs, therefore, when through organic disease or functional disturbance the formation of this ferment is wholly or even partly arrested. Another source of the glycolitic ferment is the salivary secretion.

(2) If the glycogenic function of *the liver* be interfered with materially, diabetes follows. This may result from organic hepatic disease or the fault may lie solely with the nervous system. Puncture of the floor of the fourth ventricle will also cause glycosuria, and section of the pneumogastric nerve is followed by vaso-motor paralysis of the hepatic vessels, disappearance of glycogen from the liver, and the appear-

ance of sugar in the urine. This view explains how central lesions, changes in the cord, and disturbance of the sympathetic system produce diabetes.

(3) The so-called *alimentary glycosuria* has frequently been induced experimentally by Miura and others. It results from the ingestion of more carbohydrates and peptone than can be stored in the liver as glycogen, so that some of the latter finds its way into the hepatic vessels with consecutive glycosuria.

(4) The administration of *phloridzin* produces glycosuria both in animals and man. There are two views as to the cause of phloridzin diabetes: (*a*) that the kidneys, owing to the action of the phloridzin on the renal epithelium, eliminate the sugar from the organism; (*b*) that an excessive formation of glucose occurs. The condition of the blood in phloridzin glycosuria testifies to the decomposition of proteids rather than to the mere elimination of sugar (Lépine).

(5) *The Microbic Theory*.—Paul Ernst and others have observed all forms of fungi in diabetes, thus showing that the disease is favorable to the development of various micro-organisms, but as yet none have been shown to sustain an etiologic relationship. The rapid succession of cases in a single family led Bose to suspect the contagiousness of the disease in India.

(6) Pavy's view regarding diabetes, recently advanced, is that the carbohydrates of the food are converted into fat by the protoplasmic action of cells in the intestinal villi, and enter the system in the same way as do fats taken as such. The surplus carbohydrates that escape the action of the cells of the villi are transmuted into glycogen in the liver. The glycogen stored in the liver obviously forms fat also, since this organ has some fat-forming function, and there are thus two barriers preventing the carbohydrate matter entering into the blood, and if either is deranged diabetes may result.

It is obvious that no single view explains all the cases of diabetes, and that the glycogen derived from the carbohydrates and proteids of the food (found normally in the liver and in the muscles) may be present in increased proportion, giving rise to glykemia, or be eliminated from the system on the other hand, owing to a great variety of morbid influences.

**Pathology.**—The **pancreas** in more than one-half the instances shows morbid changes. The most frequent lesion is the granular atrophy of Hanseman, occurring in 36 out of 40 cases of pancreatic diabetes. Fibroid induration of the pancreas is sometimes observed in diabetics, and is due to syphilis. Diffuse cancer of the organ may lead to diabetes, but not readily, Hanseman having observed 2 cases, and I also 2, without diabetes. Calculus, with atrophy, may or may not be associated with diabetes. Occlusion of the pancreatic duct, atrophy from pressure, and cystic degeneration of the organ may sometimes be combined with the disease. Acute necrosis of the organ may rarely lead to glycosuria, but of 100 cases collected by Fitz and Seitz only 2 had diabetes.

The **liver** is often enlarged and the seat of fatty degeneration. According to French writers, there is a diabetic cirrhosis of the organ (*cirrrose pigmentaire*), the pigment being derived from destroyed blood-cells. I have observed an instance in which diabetes coincided with tertiary syphilis affecting the liver.

**The Kidneys.**—There may be noted a benign, often intermittent, form of albuminuria, and a well-marked nephritis, with fatty degeneration, is often present. The tubal epithelium may show a hyaline change, and the lesions of acute nephritis, which may be the cause of diabetic coma, may be present.

**Nervous System.**—In rare instances organic disease of the medulla (tumors, sclerosis, etc.) is found. Cysts have been met with in the white matter of the cerebrum, and perivascular changes have also been described (Osler). Changes in the posterior columns of the cord have been noted, and a peripheral neuritis, simple or multiple, is commonly though not constantly seen. The so-called *diabetic tabes* is generally supposed to be due to multiple neuritis. Fraser and Bruce post-mortemized a case of diabetic neuritis which showed a zone of degeneration in the optic nerve. Sclerosis and enlargement of the ganglia of the sympathetic system have been noted in a few cases.

**The Lungs.**—The commonest lesions in the lungs are gangrene following pneumonia (particularly broncho-pneumonia) and the so-called diabetic phthisis. Fatty emboli have been found in the pulmonary vessels.

**The Heart.**—Arterio-sclerosis with cardiac hypertrophy is often met with, but does not constitute a peculiar lesion. Marie found pigmentary degeneration of the muscular fibers and sclerosis of the pigmented fibrous tissue.

**The Skin.**—Cutaneous pigmentation, brown or even red-black, and more or less uniform, has been reported in 9 cases (Hanot and Chauffard). It is associated with hypertrophic cirrhosis of the liver.

**The Stomach.**—Dilatation and, according to Jacobson, marked catarrhal changes are common in the early stage.

**The Blood.**—The normal proportion of sugar in the blood (0.15 per cent.) is usually increased, though there is no immediate connection between the percentage of sugar in the blood and in the urine in diabetes. Both in experimental and pathologic diabetes hyperglykemia may be marked, with moderate or slight glycosuria, and Lépine has shown that diuretics diminish hyperglykemia by increasing the glycosuria. The blood-plasma contains much fat. It is probable that the albuminoid matters in the blood may produce glucose. Glycogen probably exists in the blood-corpuscles, and not in the plasma, "where it would be destroyed by the diastasic ferment" (Dastre), and it is a normal element of the blood, apparently belonging to the leukocytes (Huppert and Czerny). The corpuscles show no special alterations.

**General Etiology.**—(a) *Heredity* is generally believed to exert a predisposing influence, since cases are observed to succeed one another in the same family. (b) *Season* also exerts an influence, diabetes appearing more frequently in the months of March, April, July, and November (Davis). (c) *The male sex* suffers much more frequently than the female. Wegeli, however, found in 107 cases that children of both sexes were affected in an equal proportion. (d) *Age.*—Most cases occur between twenty-five and fifty years. Infantile diabetes is rare, and occurs most frequently about the age of five, though it has been met with under one year. (e) *The Hebrew race* is especially susceptible. (f) *The better classes* of society furnish most instances, and particularly



that large element composed of neurotic subjects. (g) *A nervous shock* or strain or prolonged mental anxiety acts as a predisposing cause. (h) *Occupation*.—The urine of 607 individuals engaged in manual labor that required great muscular and respiratory activity showed no sugar in any case; while the urine of 100 individuals engaged in intellectual work of a more or less fatiguing character, but always intense and sedentary, showed sugar in 10 of the cases in varying proportions (Worms). (i) *Obesity* predisposes, though usually to the lipogenic form, which is generally a mild variety. (j) *Certain chronic diseases*—*e. g.* syphilis, malaria, gout—predispose. (k) *Pregnancy* has a slight though decisive influence. (l) It sometimes follows *acute infectious diseases*. (m) *Locality*.—Unlike gout, which has been increasing in recent times, diabetes mellitus has been decreasing in America. On the other hand, in certain other countries, particularly in France, diabetics appear to be constantly increasing in number, the mortality in Paris having more than doubled from 1883 to 1892, inclusive. The disease is much more frequent in cities than in rural districts.

**Special Etiology.**—Under this head may be arranged the following groups of cases: (1) *Diabetes* due to *pancreatic disease*. (2) Cases occasioned by *hepatic disease (organic and functional)*. (3) Those comparatively rare instances caused by *disease of the brain (tumors, sclerosis, or irritative lesions of the diabetic center) and spinal cord*. (4) Diabetes follows *traumatism*, and especially injuries to the head. Not infrequently it occurs after injuries to other parts of the body, such as the spine, sacral region, abdomen, etc. In 212 cases of traumatism of the head Higgins and Ogden found 20 cases of glycosuria, though only a small proportion of the cases (2) exhibited a permanent glycosuria from the date of injury. Ebstein,<sup>1</sup> after an exhaustive study of 6 of his own cases and of 44 gathered from literature, concludes that there can be no question of the direct causal relation of traumatic neurosis and diabetes. Cases of diabetes may follow injury without cerebro-spinal lesions.

**Clinical History.**—For the sake of accuracy and convenience of description the cases will be divided into the acute and chronic forms, brief reference being made to special varieties based upon an etiologic classification under Clinical Types.

1. **Acute Diabetes Mellitus.**—The instances are few and the course is, as a rule, rather subacute than acute, manifesting a predilection for the young and middle-aged. The onset is more abrupt than in the chronic form, but the characteristic features do not differ from those of the latter, more common variety. Many of the cases due to pancreatic disease are of this class.

2. **Chronic Diabetes.**—The symptoms are evolved slowly and gradually, as a rule, and prominent among prodromal conditions is dyspepsia or chronic gastric catarrh. We may also note certain nervous disorders, such as headache, mental irritability, moroseness, and insomnia, with or without gastro-intestinal symptoms. In some cases the patient suffers merely from general debility and malaise, and either frequent micturition, polyuria, or unnatural thirst is apt to be noticed. Rarely, diabetes has an abrupt onset, as after an injury or a sudden severe nervous shock. With the development of the affection the polyuria becomes

<sup>1</sup> *Deutsche Arch. f. klin. Med.*, April, 1895.

marked, as a rule, the thirst great, the appetite keen, and glycosuria appears. In spite of the enormous quantities of food taken, progressive emaciation and debility attend.

**Leading Symptoms and Complications in Detail.**—(1) *The Urinary Symptoms.*—The daily amount of urine varies from four or five pints to as many gallons. In very mild cases and in intercurrent febrile attacks it may be slightly, if at all, increased in quantity. Its color is pale, and its specific gravity ranges from 1020 to 1050, rarely being as low as 1015; it has an acid reaction, a sweetish, aromatic odor, and a distinctly sweetish taste. Sugar is present, the amount varying from  $\frac{1}{2}$  of 1 per cent. to 2 per cent. in mild cases, to 5 or even 10 per cent. in severe attacks. The total amount eliminated in the twenty-four hours varies from five or ten ounces to a pound or more. (For the most satisfactory tests for glucose in the urine *vide* Glycosuria, Diseases of the Kidney.)

Other forms of sugar than glucose (inosite and levulose) may be contained in the urine, and glycogen has rarely been found. The urine may also contain fermentation-products (acetone, alcohol, and diacetic acid). Acetone strikes a Burgundy-red color on the addition of the chlorid of iron (*vide* Acetonuria, Diseases of the Kidney).

The *urea* is greatly increased, Kaufman finding the quantity in the blood of diabetic dogs to be doubled. *Uric acid* is either normal in quantity or slightly diminished, but a large amount of ammonium is present, indicating an increase of organic acids. The phosphates may also be present in greatly increased proportion (Ralfe), and in such cases the glycosuria may be more or less intermittent. This has been described as a special variety—phosphatic diabetes. Lipuria may be present.

*Slight albuminuria*, often with an intermittent tendency, is common even in the early stages, and is not of grave significance. Well-marked nephritis with its characteristic phenomena may develop, though usually in advanced diabetes; and if albuminuria be marked, the amount of sugar excreted may be considerably diminished. The development of chronic interstitial nephritis, however, is not a favorable complication, as some have supposed. With or without nephritis, arterio-sclerosis may be observed, and pyelo-nephritis (rarely) and cystitis (not rarely) may appear as complications. As the result of fermentative processes in the bladder gases may form (*pneumaturia*). Impotence is a not infrequent, and often an early and a very significant, symptom; later this condition may improve spontaneously.

(2) *Digestive Symptoms.*—Although a general feature, thirst may be discussed under this head. This symptom may be most distressing, necessitating the drinking of large quantities of water at frequent intervals both by night and by day. The amount of water taken stands in direct relation to the amount eliminated, though not necessarily to the daily amount of sugar excreted. Notwithstanding the fact that the increased amount of water is needed to dissolve the sugar, cases of confirmed diabetes are met with in which thirst is not marked and the amount of urine passed but little above the normal. Cases are also encountered in which the amount of urine is large and the percentage of sugar excreted very low. The cause of the unusual thirst is not



quite clear, though it is probable that the chief factor is the increased systemic demand for liquids.

The appetite is abnormally large and sometimes almost insatiable (bulimia), and there may be an intense craving for carbohydrates. I have, however, met with two instances of well-developed diabetes in which the appetite was not inordinate. The cause of the ravenous appetite is probably to be found in the defective assimilative processes. Considering the quantity of food consumed, the digestion is often surprisingly good, but the association of dyspepsia and diabetes is by no means an uncommon one. As a rule, there is constipation, though brief intervening attacks of diarrhea may occur.

The tongue is generally dry, large, often presenting a rough and fissured surface, and it may either be coated or red and glazed. The gums sometimes swell, and may ooze blood. The saliva is scanty and its reaction persistently acid, while the salivary secretion may show sugar on testing. The teeth decay, and aphthous stomatitis or thrush may attack the oral cavity.

The liver is frequently somewhat enlarged, though the biliary secretion usually is not disturbed; jaundice may, however, arise as a complication. Marie has given a description of *pigmentary "hypertrophic cirrhosis with diabetes mellitus,"* of which only 9 undoubted cases have been published. It appears late in adult life, and, in addition to the symptoms of diabetes mellitus, slight ascites, considerable hypertrophy of the liver and spleen, with brown or even gray-black cutaneous pigmentation, are among the chief features noted. There is no true icterus as a rule, but the urine is highly colored and contains bile-pigments.

(3) *Cutaneous Manifestations*.—Diabetic urine, on account of the sugar it contains, has irritant properties, and often produces in the female pruritus vulvæ, a most troublesome symptom and one that should always excite suspicion of this disease. In the male, balanitis often occurs, due to the effect of the decomposing urine, and from the same cause the genitals and adjacent cutaneous surfaces may be the seat of eczema. This seems to be more common in women than in men. The skin is usually harsh and dry, though rarely copious perspiration may be observed, and particularly if phthisis be a complication. The hair often falls off, and in one case in my own practice shedding of the nails occurred. Among the commonest of the early cutaneous symptoms are furuncles and boils. Later large carbuncles often appear. Gangrene (especially of the feet) due to arterio-sclerosis is not infrequent, and edema, arising independently of nephritis, is not uncommon.

(4) *Nervous Symptoms*.—Diabetic coma is the most important as it is the most grave symptom, marking a fatal termination in more than half the cases. It is of most frequent occurrence in instances showing rapid wasting, and is heralded by a fruity odor in the exhaled breath and in the urine. The polyuria and glycosuria lessen, while acetonuria increases as a rule. The cases may be arranged into the following clinical groups:

Group 1. To this belong abortive forms that terminate in quick recovery. This process may be repeated several times at intervals, and at last a fatal coma may supervene.

Group 2. Perhaps the largest group, in which the diabetic coma fol-



lows some form of exhausting exercise. It may end fatally in a few hours or, though less frequently, in three or four days.

Group 3. This is a comparatively small class, and is characterized by collapse of the circulation (small, rapid, feeble pulse, cyanosis, etc.), leading to coma. It is induced either by over-exercise or by intoxication. I have seen 2 typical instances, but feel that it may be questioned whether most of these cases should be classed as diabetic coma.

Group 4. Without previous dyspnea or distress there appear such symptoms as headache and signs of intoxication, and these are followed quickly by deep and fatal coma (Frerichs).

Group 5. Here diabetic coma is preluded by symptoms of some localized disorder, such as gastro-enteritis, pharyngitis, pneumonia, gangrene, or carbuncle. The attack sets in with headache, delirium, distress, and dyspnea both inspiratory and expiratory. Cyanosis may develop early, and, if so, cardiac failure precedes the coma. The duration is from one to five days. This group, which was first described by Frerichs, may have a different onset, and I have seen two cases, one attended by carbuncle, the other with gastric symptoms, in which headache, dyspnea, and great distress were conspicuous by their absence. The coma ended in a speedy death.

Group 6. Hirschfeld has recently described a class of cases in which we find, in old persons, a moderate glycosuria and coma supervening under the influence of gangrene or carbuncle.

The *causes* of diabetic coma are still obscure. Hirschfeld points to insufficient nutrition from an exclusive meat diet as an important factor. Kussmaul believed diabetes to be due to acetone. Klemperer, after a careful study of 21 cases, concludes, to his own satisfaction, that the condition is not due to an acid-intoxication, and that there probably exists in the blood a toxic substance which produces an increase of acidity ("acetonemia") and coma. In this connection the fact that an increased destruction of nitrogenous material may be the cause of fatal coma, not only in diabetes, but also in other complaints (*e. g.* pernicious anemia), must be recollected. Again, from the character of many preliminary symptoms diabetic coma must sometimes be of uremic origin, while those cases that follow suppuration and gangrene may be septic in nature.

*Peripheral neuritis* is common. The most frequent form is diabetic tabes, indicated by an absence of the knee-jerks, darting pains, paresis of the extensors of the foot, and by the peculiar gait (steppage). Other symptoms pointing to neuritis may be numbness, tingling, and certain trophic disturbances, such as shedding of the nails and perforating ulcer of the foot. Neuralgia may be a troublesome symptom, particularly when it is of the symmetrical sciatic type, and it points to neuritis. The same is true of paraplegia, a condition that is sometimes observed.

*Psychopathia* (*e. g.* irritability of temper, hypochondriasis) may sometimes be present, and temporary hemiplegia has been noted.

(5) *Special-sense Symptoms*.—Not infrequently cataract develops, leading to blindness. Its cause is not clear. Transient ptosis and strabismus may also appear, and among other ocular conditions are optic-nerve atrophy, retinitis (often due to associated nephritis), and

hemorrhage. Amaurosis is rarely observed. Among the aural symptoms I would mention otalgia, otitis media, and mastoid disease.

(6) *Muscular Symptoms*.—In diabetics there is a tendency to cramps, especially in the calf of the leg, that does not show itself during the day, but appears during the night and on waking in the morning. Unschuld found it present in 33 out of 109 cases. Another variety of cramps that may appear at any hour of the day has been noted in connection with the so-called "gastric crisis," which may be due to cramps affecting the diaphragm. In these attacks colicky pain in the epigastrium, with vomiting, and febrile reaction attend.

(7) *Respiratory System*.—Serious pulmonary complications not infrequently appear in the advanced stages, and often cause death. The most frequent is pulmonary tuberculosis, which has the customary termination, and does not differ from the usual form of the disease. A second, quite frequent complication is gangrene, which may either be limited to circumscribed foci or form a general condition. The peculiar and highly offensive odor of the expectoration that is so characteristic of gangrene may be wanting here. A very serious type of secondary pneumonia sometimes occurs, and may terminate in gangrene.

(8) *Circulatory System*.—The pulse may be of natural frequency and tension. In other cases it is somewhat slow, and the tension may be increased. This is often due to an associated arterio-sclerosis, particularly when gout plays a prominent rôle in the etiology. The heart is sometimes quite weak, as shown by the visible first sound and the feeble, small, irregular, or intermittent pulse. As to frequency, the pulse varies greatly: it may be slow (brachycardia), not exceeding 40 or 50 beats per minute, or it may, though more rarely, be accelerated, reaching 120 or even 130 beats. Dyspnea, a tendency to syncope, and gastric disturbance may be seen in combination. Cardiac failure, leading to coma and speedy death, has already been mentioned.

(9) *Constitutional Symptoms*.—Usually there is a constantly increasing loss of flesh and strength. In the mildest types, however, and particularly if they occur in the latter half of life, good bodily nutrition and a fair degree of strength may be maintained indefinitely. When emaciation is progressive the polyuria is apt to be proportional. The temperature is at first normal, later usually subnormal, though intercurrent febrile attacks, due to complications, are often witnessed. Finally, as the result of profound emaciation and weakness, the patient betakes himself to bed.

**Clinical Varieties.**—(a) *Infantile Diabetes*.—Heredity, traumatism, and convalescence from severe acute infectious diseases are the most potent causes. The type is severer and the course shorter than in adults, and the latter may be quite acute. On the other hand, a comparatively mild chronic form is, though rarely, met with in children.

(b) *Pancreatic Diabetes*.—This is a grave variety, and sometimes presents, in addition to the usual features already pointed out, evidences of pancreatic involvement. There may be epigastric pain: the fats are poorly assimilated; and the physical signs may rarely point to pancreatic growth. Marked polyuria and great thirst may be absent, and albuminuria is rare. It is to be recollected, moreover, that not all cases of pancreatic disease are attended with diabetes.



(c) **Alimentary or Lipogenic Glycosuria.**—This is caused by dietetic errors, and especially by excesses in eating and drinking, combined with physical inactivity. Bloch experimented on 50 patients, and found that the amount of grape-sugar that could be given before glycosuria appeared differed widely in different diseases. Frequently the smallest quantity was required in nervous diseases, and particularly in cerebral affections. Von Jaksch induced alimentary glycosuria in cases of hysteria and in cases of phosphorus-poisoning with fatty degeneration of the liver. This form of the disease is often a temporary affair, and restoration to health from the primary affection may defy further attempts at the production of glycosuria. It is a mild form of the disorder frequently observed among persons inclined to obesity, and especially among those who live luxuriously and indulge too freely in rich dishes. The percentage of sugar in the urine is usually small, while polyuria and polydipsia are moderate.

**Prognosis.**—In acute diabetes the duration varies from a few days to eight or ten weeks, while in chronic diabetes the course ranges from one or two to five or even ten years. When the disease commences in the declining period of life, the course is longer still, and has been known to reach fifteen years. The shortest limit is found in infantile diabetes. The outlook is greatly influenced by the type of the individual case. The severe forms are generally fatal, and are often rapid in their course, occurring, as a rule, at an early period of life and in persons with an hereditary taint. They are not infrequently due to pancreatic disease. The mild types and those that occur later in life offer a more hopeful prognosis, and in certain cases the withdrawal of all carbohydrates from the diet will cause the sugar to disappear from the urine. Of the special varieties, *alimentary glycosuria* is altogether favorable in its course, *traumatic diabetes* somewhat less so, while the prognosis of the *pancreatic form* is quite unfavorable.

On the whole, the later in life the disease begins the more favorable the prognosis. Stout persons also bear saccharine diabetes better than lean. Pre-existing affections may render the prospect quite gloomy, and the coincidence of certain symptoms and complications indicates grave danger; among these are coma, phthisis, gangrene, pneumonia, cardiac weakness, and nephritis. Of 108 such cases, 64 per cent. terminated fatally (Wegeli), and between the ages of four and five years—the most dangerous period—20 out of 29 cases perished.

**Diagnosis.**—Diabetes is distinguishable by means of (1) its causal influences and its pathologic antecedents and relations; (2) its gradual onset, often marked by certain suspicious symptoms (*e. g.* debility, impotence, symmetrical sciatica, cataract, furunculosis); (3) the persistent (rarely intermittent) presence of glycosuria, polyuria, and, later, acetoneuria and albuminuria; (4) the inordinate thirst and appetite; (5) cutaneous boils, carbuncles, gangrene, pruritus vulvæ in the female, balanitis in the male; (6) neuritis (especially double sciatica), diabetic tabes, and coma; (7) muscular cramps; (8) special complications; and (9) the long course with slowly progressive asthenia and wasting.

In suspicious cases, even before the discovery of sugar in the urine, particularly if adiposity should develop, grape-sugar may be administered for diagnostic purposes. If glycosuria result, the cases are to be



treated just as in pure diabetes. Transient glycosuria, however, should be differentiated from the genuine affection.

**Treatment.**—1. A properly regulated diet is of the first importance. Such food-articles as contain starch or sugar (honey, sugar, ordinary flour or bread, biscuits, rusks, toast, arrow-root, oatmeal, cracked wheat, potatoes, tapioca, sago, peas, beans, turnips, carrots, parsnips, asparagus, artichokes, squashes, beets, corn, rice, hominy, the stalks and white parts of cabbage, lettuce, broccoli, figs, grapes, prunes, apples, pears, bananas, jams, syrups, sweet pickles, chocolate, cocoa, liquors, and especially sweet wines) are either to be altogether prohibited or restricted to definite quantities, as will be pointed out below. Among articles to be forbidden are also the livers of animals, mollusks (oysters, etc.), and the inside meat of crabs and lobsters. The chief diet must be animal, since the non-nitrogenous substances are to a very limited extent, and in some instances not at all, assimilated. My own plan is to first note the effect of a rigid dietary as follows:

(a) *Animal food*: Fresh meats, poultry, game, bacon, ham, fish of all kinds, including crabs and lobsters (except the inside meat of the latter). Fatty substances in large quantities (3vij—256.0—daily), with a view to restricting nitrogenous destruction, are highly commended by Klemperer, and the free use of butter is also urged, eggs, cream-cheese, curds, and buttermilk being allowed.

(b) *Vegetables*: Saur-kraut, lettuce, sorrel, mushrooms, water-cresses, spinach, chicory, celery, cucumbers, mustard-cress, and pickles of various sorts (except sweet).

(c) *Bread*: The crust of a French roll, first recommended by Flint.

(d) *Fruits*: Lemons, oranges, and nuts (except chestnuts).

(e) *Beverages*: Milk enough for cooking purposes; tea and coffee, sweetened with glycerin or saccharin; alkaline mineral waters (Saratoga-Vichy, Seltzer-water), simple water with a slight amount of brandy, and acidulated drinks. Also Bass's ale, in which all the sugar is converted into carbonic acid and alcohol.

This strict diet usually causes the sugar to diminish greatly in amount, and in many cases to disappear entirely. If the patient keeps well nourished and strong, it may be continued until recovery. On the other hand, nothing is gained by relieving the glycosuria and polyuria if it be accomplished at the expense of the general strength of the patient. This latter result is sometimes witnessed, and in such cases carbohydrates in limited amounts are indicated. S. Solis Cohen, after repeated trials, recommends levulose as a form of sugar that can be assimilated without augmenting the excretion of glucose. With lean patients he uses 3j (32.0) per day; with stout persons, only enough to act as a sweetening agent. Lactose has been found to give similar results. Hale also strongly urges levulose, after considerable personal experience. As a substitute for the latter agents a small amount of ordinary bread or potatoes, as the patient prefers, may be allowed. The effects upon the general condition of the patient, as well as upon the glycosuria (ascertained by a daily quantitative estimation of the sugar in the urine), are to be carefully noted, and the proportion of carbohydrates may be increased gradually until the limit of the system's ability to assimilate them is found. A more generous dietary is allowable only

after the sugar has been absent from the urine for a couple of months, and then it is to be adopted in a gradual manner. A skimmed-milk diet has been recommended by Donkin, Tyson, and others, but I have found it to be vastly inferior to the one above laid down, and hence it should not be tried unless the latter cannot be taken.

2. Next to an appropriate diet stand certain directions as to **proper hygienic living**: (*a*) All forms of mental excitement and worry must be avoided; (*b*) moderate and regular physical exercise aids metabolism, and is thus directly useful; massage may be substituted for active exercise when the latter is prohibited on account of weakness; (*c*) the diabetic requires a temperate and equable climate; (*d*) a daily tepid bath if the patient be feeble, and a cold bath if he be strong, are to be commended; (*e*) flannels should be worn next to the skin all the year round; (*f*) the living and sleeping apartments must be thoroughly ventilated; (*g*) the teeth must receive careful attention in order to prevent caries.

3. The **medicinal measures** deserve only third place in the treatment of diabetes, and of these opium is still the chief. It is not necessary to employ it in all cases, but it may be tried if the dietetic and hygienic treatment before recommended cannot be carried forward or fails to effect a cure. Opium seems not only to exert an influence over the polyuria and the excretion of sugar, but it almost invariably lessens the intense thirst and conduces to refreshing sleep. The drug is well tolerated by diabetics. The commencing dose may be gr. j (0.0648) three times daily, and later increased to gr. v (0.324) or even to gr. x (0.648) three times daily. If morphin be employed, we may begin with gr.  $\frac{1}{4}$  (0.0162) and increase the dose to gr. j (0.0648) or more three times daily. Pavy warmly advocates the use of codein (gr.  $\frac{1}{2}$ — $\text{ij}$ —0.0324—0.1944, three times a day). My own best results have been obtained from the use of the latter remedy in the form of the sulphate, in ascending doses, commencing with gr.  $\frac{1}{4}$  (0.0162) three times a day, and augmenting the dose by gr.  $\frac{1}{4}$  (0.0162) every second day until gr.  $\text{ij}$ —0.129 (rarely more) are taken thrice daily. Codein possesses the advantage of being less constipating and less likely to disturb the digestive function than either opium or morphin. In patients of a full habit the alkaline waters exercise a valuable influence; Bethesda, Carlsbad, and Vichy of France have long had a reputation. For the foreign water our native alkaline waters may be substituted, especially the Saratoga-Vichy. While these are valuable adjuncts, they are, however, without the curative and specific effect that is claimed for them by certain authorities.

Among other therapeutic agents that have been employed are the following: the solution of the bromid of arsenic,  $\text{Mij}$ — $\text{v}$  (0.199—0.333) three times a day, after meals—in some cases a useful adjuvant to the treatment above outlined; potassium bromid, gr. xx (1.296), three times a day, approximating in efficacy the latter remedy; guaiacol,  $\text{Mv}$ — $\text{x}$  (0.333—0.666), three times a day in a tablespoonful of milk or cod-liver oil, has given excellent results (Clemens); antipyrin (gr. x—0.648, three times a day); sodium salicylate, gr. xv (0.972), three times daily, lessens the formation of sugar; and strychnin, gr.  $\frac{1}{30}$  (0.0021), three times daily, is an almost invariably useful remedy. Of the numerous remedies in whose favor convincing evidence is wanting, but which are employed by different clinicians, the following may merely be enu-



rated: Fowler's solution, potassium iodid, iodoform, lactic acid, glycerin, nitroglycerin, creasote, quinin, jambul, lithium, and ammonium salts.

The treatment of diabetes by *fresh pancreas* or by dry or glycerin extracts has been uniformly unsuccessful. These preparations have been employed to supply the ferment (internal secretion) essential to the assimilation of sugar. R. Lépine has obtained from the fresh pancreas, from saliva, and from the diastase of malt a glycolitic ferment by a method which, he tells us, still requires to be perfected. This agent he has used in 4 cases of diabetes with a fair degree of success. Says Lépine: "I have never thought of treating cases exclusively by the glycolitic ferment, which responds only to an indication—diminished glycolysis—which I have noted in diabetic patients, but there are other essential elements of the disease that must also be treated if good results be desired." Williams tried grafting sheep's pancreas in diabetics in two cases, and, while the grafts were found to have taken hold, the results, so far as pertained to the course of the complaint, were unsatisfactory.

**4. Symptomatic Treatment.**—Most symptoms demanding therapeutic interference the competent physician is prepared to meet by following general rules. The management of diabetic coma, however, will be briefly alluded to, though, unhappily, the agent is yet unknown that is capable of controlling this dangerous complication. Without stopping to mention all of the remedies that have been employed, it is premised that so long as we are ignorant of the cause of coma all efforts at cure must be regarded as being purely empirical. When it was thought to be due to acid-intoxication, alkalies were employed in its treatment, and Klemperer gave large amounts of alkalies in 9 cases by intravenous injection, without preventing a fatal issue. As previously intimated, Klemperer urges the use of fatty substances in large quantities as the best means of restricting nitrogenous destruction, and thus preventing the condition to which diabetics so frequently succumb. When a disgust develops for fats a substitution-method of treatment consists in administering alcohol (3iss—48.0—per day). Like carbohydrates, alcohol in small quantity, and in small quantity only, checks waste, and, according to Hirschfeld, may lead to an accumulation of flesh. Strychnin, digitalis, or ether may be tried hypodermically during the attack. Prolonged tepid baths with occasional douching have seemed to produce beneficial results in some cases, and are worthy of a trial in all.

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## DIABETES INSIPIDUS.

**Definition.**—A chronic nervous affection, characterized by constant thirst and an excessive flow of urine, which, however, is free from sugar and albumin and is of low specific gravity.

**Pathology.**—No definite or characteristic lesions have been noted, though some degree of enlargement of the kidneys, together with sacculation, due to pressure backward upon the renal structure by the enormous quantities of urine in the bladder and ureters, has been observed. The ureters and pelves of the kidneys may be dilated, and the bladder,



owing to constant over-distention, may be hypertrophied. The nervous lesions are diversified, but are not peculiar to simple polyuria. Most important, perhaps, are the tuberculous and other tumors about the floor of the fourth ventricle.

**Etiology.**—(a) Diabetes insipidus is often induced by *nervous influences*—shock, fright, etc.—and may also be of traumatic origin. In the majority of the latter cases it follows injuries to the head, but also, more rarely, it may be traced back to injuries of other parts of the body. Tuberculous and other lesions in the vicinity of the floor of the fourth ventricle may produce polyuria. It has also been caused by paralysis of the sixth nerve, with or without meningitis. (b) It may occur during convalescence from *acute infectious diseases*. I have seen 2 instances after influenza in young subjects. (c) *Intemperance*, especially the consumption of inordinate quantities of malt liquors, proves a cause. In several of my own cases the amount of urine passed was out of all proportion to the quantity of fluid ingested. One of these patients consumed three pints of beer daily, while the urine excreted amounted to eight quarts. (d) *Heredity*.—Weil found in four generations of a certain family consisting of 91 members, that 23 exhibited continuous polyuria—all, however, remaining in good health. (e) *Age*.—The disease is relatively more frequent in childhood and early adolescence than is diabetes mellitus. Of 70 cases collected, 22 were under ten years of age, and 13 between ten and twenty (Roberts). Diabetes insipidus may be *congenital*. (f) The great proportion of cases occur in males as compared with females.

**Nature of the Affection.**—The specific cause of the disease, if it have one, is as yet undiscovered. We are totally ignorant of its true nature, though the facts discovered by Bernard, that either a puncture at a certain spot in the floor of the fourth ventricle or section of the vagus causes polyuria, go to show that it is of nervous origin. It is true that the disease may come on in persons apparently in robust health without discernible causative agencies. In many instances, such as organic affections of the brain or abdominal tumors, the condition is purely symptomatic, and these are probably not to be classed as cases of genuine diabetes insipidus, which is a vaso-motor neurosis, usually of central, though sometimes of reflex, origin.

**Clinical Symptoms.**—The onset is gradual, as a rule, but when it follows a fright or traumatism it may develop quickly. There are two main symptoms—the passage of an enormous quantity of limpid urine, and the constant thirst. The daily amount of urine varies from 20 to 60 pints (10–30 liters); it is transparent, and the specific gravity is low (1001 to 1005). While the percentage of solids is lessened, the total is usually about normal, and may even be increased. Albumin and sugar are rare, but in a few cases inosite has been detected. The act of micturition is of very frequent occurrence, and the quantity of urine passed at each sitting surprisingly large. The persistent thirst necessitates frequent drinking, but the voracious appetite seen in diabetes mellitus does not mark this disease, in which the appetite is only slightly increased. As a result of the polyuria the skin and mucous membranes are abnormally dry, as in genuine diabetes. But, unlike the latter affection, a fair degree of bodily nutrition is maintained as a rule.

The saliva and other digestive secretions are scanty, and this, together with the good appetite, is a fact which explains the disturbances of digestion sometimes met with. The tolerance of the system to alcohol is often phenomenal. Associated nervous phenomena are frequently observed, such as neurasthenic symptoms, insomnia, and chorea.

**Prognosis.**—The majority of instances proceed to recovery sooner or later, while others pursue an almost endless course—forty or even fifty years in duration—and the patient meanwhile retains his general good health. There is a small group of grave cases that are due to organic diseases either of the brain or abdominal organs. Death may also be occasioned by some intercurrent complicating condition.

**Diagnosis.**—The clinical recognition of diabetes insipidus rests upon—(a) the enormous amount of urine passed; (b) its low specific gravity; and (c) the absence of sugar and albumin.

**Differential Diagnosis.**—Among affections that must be differentiated are *diabetes mellitus*, which has a single point of resemblance—namely, the polyuria; *hysterical polyuria*, which is transient and accompanied by other hysterical manifestations; and *chronic interstitial nephritis*, which sometimes distinguishes itself by the presence of albumin and hyaline casts in the urine, arterio-sclerosis, and cardiac hypertrophy.

**Treatment.**—The amount of drinking-water is to be moderated in a gradual, cautious manner. To insist upon a sudden great reduction is productive of harmful effects, but the patient should be warned not to exceed his actual necessities. I also find that methodic physical exercise acts very beneficially.

Of medicines, nervines, especially valerian and its preparations, are useful in the idiopathic variety of the complaint, and may be given in the form of the ammoniated elixir (3j-ij—4.0–8.0) three or four times daily. The valerianate of zinc, quinin, and iron may be variously combined, according to the indications presented by special cases. Ergot and gallic acid have long enjoyed a high reputation in this disease. The commencing doses should be moderate, and then be increased until full physiological doses are employed, this method often bringing about admirable results. Antipyrin, acetanilid, the bromids, and arsenic have been extensively employed and lauded by different writers in the treatment of this affection. My own best results have been attained by the use of ergot. Next to this agent the bromids and acetanilid, given alternately at intervals of a couple of weeks, have been found to be most useful. If a primary disease exists, it must be met on intelligent general therapeutic principles.

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## ARTHRITIS DEFORMANS.

(*Rheumatoid Arthritis; Rheumatic Gout.*)

**Definition.**—A chronic disease, characterized by progressive changes in the arthritic structures (cartilages, synovial membranes, etc.) and by osseous periarticular formations, producing great deformity. The affection may rarely be acute in its course.

**Pathology.**—It is here that the identity of the disease is distinguishable. Among early gross changes there may be an effusion into the affected joints, but this disappears later. The cartilages are absorbed, the process beginning centrally, where there are both the maximum amount of friction between the opposed cartilaginous surfaces and the minimum blood-supply. Disappearance of the cartilages is naturally followed by contact of the ends of the bones, the latter becoming polished and resembling ivory as the result (eburnated). The degenerative process, which is, as in the case of the cartilages, due to friction between the bony extremities, may lead to absorption of the latter with a consequent alteration of shape.

At the periphery, where pressure is slight or even absent, the cartilages become greatly thickened in consequence of persistent irritation, and later become ossified, forming osteophytes which overlie the articular surfaces. These may lock the joints, though perhaps oftener they cause partial ankylosis. Bony nodules may also be formed from the periosteum of the shafts of the bones.

Almost simultaneously the *synovial membranes* become inflamed, a proliferation of their cells taking place, and this exudate may undergo organization and rarely ossification. Later the *capsule* and the *ligaments* are thickened, causing a restriction of movement of the affected joints and producing pseudo-ankylosis. Less frequently they soften and weaken to such an extent that often partial, and sometimes complete, dislocation of the joints ensues; but displacement of the ends of the bones, amounting even to complete luxation, may also be due to absorption of the heads of the bones. This is often observed in the head of the femur, producing the so-called *morbis coxæ senilis*. Muscular wasting occurs and may be profound. Local neuritis has been noted.

The *histo-pathologic* changes consist in cell-proliferation, with fibrillation and softening of the matrix of the cartilages, followed by absorption due to pressure. At the margin, however, proliferation of the cells leads to massive nodulation.

**Etiology.**—The nature of the disease is still dubious, though the old view, that it is closely connected with rheumatism on the one hand or gout on the other, should be abandoned. J. K. Mitchell long since maintained that rheumatoid arthritis is of nervous origin, being especially dependent upon affections of the spinal cord, and without stopping to adduce all of the facts that tend to support this theory, the following deserve mention: (1) Diseases of the cord (locomotor ataxia, etc.) are known to cause arthritic conditions; (2) The character of certain causal factors, such as nervous shocks, grief, etc.; (3) The symmetry of the joint-deformities; (4) The time of occurrence; and (5) Noticeable trophic disturbances that are frequently associated. Falli<sup>1</sup> autopsied 4 cases, 2 of which were typical, and in the latter lesions were found in the anterior horns of the spinal cord, atrophic in the first case, but degenerative as well as atrophic in the second. According to Falli, not all cases of arthritis deformans are to be interpreted as instances of nervous disease. Other theories, especially the mechanical, chemical, and

<sup>1</sup> *Il Policlinico*, Dec., 1894.





FIG. 31.—Hand of M. R., aged fifty years, showing characteristic deformity, including outward deflection of fingers, in advanced arthritis deformans.



microbic, have been propounded, but have not received the same measure of support as the neuro-trophic.

(a) The disease frequently follows *nervous shocks*, mental worry, and deep grief. (b) *Females* are more frequently victims than are males, the proportion, according to the statistics of Garrod, being about one to five in favor of the former sex. To account in part for its greater frequency in women is the fact that sterility and certain ovarian and uterine complaints seem to exert a strong etiologic influence. (c) *Age* exerts a decided influence. It is most frequently contracted in the third decade of life, though it has been noted as late as the end of the fifth. It occurs also in children, though rarely. Out of 307 cases treated in the Devonshire Hospital during 1892, only 2 per cent. manifested the disease before the age of ten. (d) *Heredity* has been traced in some instances, and in many a *family tendency* to joint-affection. (e) Though it occurs in all classes of society, the poor or those exposed especially to debilitating influences are more liable than the rich.

Dor claims to have succeeded in finding a definite organism in arthritis deformans. He also claims to have reproduced the disease by injecting cultures directly into the blood of rabbits, and considers the germ an "attenuated culture" of the *staphylococcus pyogenes aureus*. His observations, however, have not as yet received adequate experimental confirmation.

(1) **Symptoms of the Chronic Form.**—At first one joint, usually of the hand, is slowly involved; soon the corresponding joint on the opposite side is attacked. These may recover apparently, but are soon reinvaded and grow progressively worse. The affected joints slowly enlarge, and are moderately painful, particularly on movement. Pain, however, may either be slight or even absent, or severe (rarely agonizing) in character. There is neither redness nor tenderness, as a rule, but on palpation an effusion, variable in extent, is generally detectable. The course during the early stage is often marked by periods of improvement, alternating with exacerbations in the local symptoms, and especially in the swelling and pain. While, as intimated, one or two joints only are affected at the start, gradually those of the feet, arms, legs, and trunk are invaded symmetrically, until, in the worst cases, every joint is deformed.

The most characteristic symptom is the *deformity*, which manifests itself earliest in the hands. The fingers are generally pointed toward the ulna, rarely toward the radius, and the presence of the osteophytes and the immensely thickened capsular ligaments, together with the retracted muscles, all tend to alter entirely the shape of the joints. The fingers, for example, are flexed and extended upon the hand, and sometimes overlies one another. With the progress of the deformity a partial, and less often a complete, luxation of the joints occurs (see Fig. 31). The joints may become finally either quite fixed, owing to the presence of the periarticular osteophytes, or a limited degree of movement may remain.

*Palpation* and *auscultation* of the involved joints reveal crepitation during movement. Strangely enough, the thumb remains intact, compensating for the loss of the functional movement of the fingers to a remarkable extent. In addition, the hand is sometimes less affected



than the rest of the joints—a fact which enables the patient to perform a great variety of even delicate movements. The adjacent muscles become wasted and are the seat of contractures, causing flexion of the limbs, especially of the thigh upon the abdomen and the leg upon the thigh. Other trophic changes, such as pigmentation or glossy areas of the skin, may be observed, and in 3 of my own cases onychia was present. In extreme instances the decubitus is lateral and the patient utterly helpless.

The *course* of the disease throughout the more advanced stages is exceedingly variable. Its advance may be arrested and the general health remain unimpaired, and this may take place after implication of but a few joints, so that the entire affection may be confined to a comparatively small part of the body, either in the upper or lower extremities. In progressive cases more or less gastro-intestinal disorder arises; the symptoms of indigestion appear, the appetite is impaired, and there is constipation. The patient's sufferings make him irritable. Hypochondriasis may be a concomitant. In established cases the pulse is persistently rapid and the skin inclined to free perspiration.

**Clinical Varieties.**—(1) Of the **chronic form** there are certain subvarieties. The disease may be limited to a single joint (*monarticular*), this form most commonly affecting the hip-joint, when it is known as *morbis coxæ senilis*. It is seen generally in old men, and often follows an injury. Its features—pathologic and clinical—including the muscular wasting, are the same in kind as those of the *polyarticular* variety. Monarticular arthritis deformans may also be confined to the shoulder-joint or the knee, and, as in the preceding form, men who have passed the middle period of life are mainly affected.

A special variety, which is generally not monarticular, involves only the vertebræ (*spondylitis deformans*). This may be combined with *morbis coxæ senilis*, or the condition may be confined to the cervical spine, as in a recent case of my own, thus preventing flexion of the head. A fair degree of rotation usually remains, but it sometimes happens that the entire spinal column is involved and held in a perfectly rigid position.

Still another form in which the distal joints of the fingers become knobbed (*Heberden's nodes*) demands separate description. Heberden's nodosities occur chiefly in women between the thirtieth and fortieth years, though I have seen one case which began after the fiftieth year in a lady who had previously suffered from gout. This patient died suddenly of apoplexy two years since. According to Heberden, who first described them, the nodes have no intimate association with gout, and at least 2 cases have fallen under my notice in which the sufferers had always been free from the latter disease. At first the affected joints become swollen, tender, and painful, and then seemingly undergo great improvement. The condition, however, is progressive, advancement occurring in the form of fresh exacerbations, which are often traceable to errors in diet, and are separated by remissions in the local symptom at longer or shorter intervals. The morbid process is the same as in rheumatoid arthritis, and the destructive changes in the joints proceed until distinct hard nodules are formed. These are usually most marked at the sides of the extensor surfaces of the second phalanges. The disease does not

spread to any of the larger joints, and, although incurable, it is free from danger to life. Heberden's nodes do not, however, imply longevity, as has been erroneously supposed.

(2) **The Acute Form.**—This is comparatively rare, and occurs commonly between the ages of twenty and thirty—at an earlier period of life than the chronic form. It is more frequent in children under ten than is the latter, and is more common in women than in men. Among its common antecedents in women are pregnancy, delivery, excessive lactation, and the menopause. Multiple arthritis, affecting both the large and small joints, sets in acutely, and there are pain and either a slight redness or a considerable swelling, due chiefly to an effusion which is intra- rather than periarticular. There is only a slight tendency to migration from joint to joint, a slight febrile disturbance, and no tendency to cardiac complication.

**Differential Diagnosis.**—The diagnosis between the chronic form of the disease and *chronic rheumatism* is not always an easy one. In the latter, however, there are fewer joints involved, and usually the larger ones only are implicated, while there is an absence of the peculiar deformity and the marked fixity of the joints which are quite characteristic of the former. There is no tendency to cardiac complications in chronic rheumatoid arthritis, and the course is more progressive. A monarticular arthritis which differs in its morbid process from rheumatoid arthritis sometimes affects the shoulder-joint. It is not uncommon, and is “characterized by pain, thickening of the capsule and of the ligaments, wasting of the shoulder-girdle muscles, and sometimes by neuritis” (Osler). I have met with 5 instances of this sort, in all of which pain was intense and persistent and the course rather subacute than acute. All ended in recovery.

The acute form is frequently confounded with *acute articular rheumatism*, from which it is to be discriminated by the special etiologic factors, the less severe pain, the less marked redness, the slight tendency to migration from joint to joint, the slighter febrile disturbance, and by the practical freedom from cardiac complications. Gout will be distinguished in the description of that disease.

**Prognosis.**—Though incurable, rheumatoid arthritis is not immediately dangerous to life, and in a certain proportion of the cases improvement, and in a smaller proportion arrested progress of the disease, may be expected.

**Treatment.**—This especially involves measures that are directed toward the improvement of bodily nutrition—a generous dietary, systematic warm bathing, and an abundance of fresh air, with properly regulated physical exercise. Tonics may be necessary to invigorate the economy, and particularly iron to overcome the anemia. The prolonged use of cod-liver oil in conjunction with other remedies has given excellent results in a few instances under my care. Of special agents, the most satisfactory in their effects, and particularly if administered early in the disease, are iodine and arsenic. An eligible form of the latter is arsenious acid, given in granules (gr.  $\frac{1}{36}$ —0.0018, after each meal), while the former may be administered in the form of a saturated solution of sodium iodid, of which ten to fifteen drops may be given in milk before meal-time. The patient may be sent to a warm climate in winter and to a

cooler one, preferably a mountain resort, in summer. These patients also do well at certain mineral springs, such as the sulphur springs of Virginia, the hot springs of Arkansas or Töplitz, at Baden in Switzerland, and the warm sodium chlorid baths in Wiesbaden. Hot mineral spas should only be resorted to in the early period of the affection. Striimpell has seen excellent results follow the employment of hot sand-baths, which can be used at home; the result is, as a rule, especially favorable when the extremities alone are involved.

*The local means* are of the highest value. If the joints be inflamed, cold compresses, covered with oiled silk, to which some narcotic agent may be added, will afford relief. This should be followed by thorough and systematic massage, which is our best measure for the reduction of the swelling (by promoting absorption of the inflammatory exudate) and for lessening joint-rigidity. It also restores the atrophied muscles and assists the general health. Swedish movements are useful in maintaining mobility and often in restoring that which has been lost.

## GOUT.

(*Podagra.*)

**Definition.**—A form of perverted nutrition due to an auto-infection, accompanied by the formation of a variable (usually increased) amount of uric acid, and characterized clinically by attacks of acute arthritis, with or without uratic deposits in and around the joints.

**Nature of the Affection.**—The numerous theories that prevail at present in regard to the disease are irreconcilable, but it seems certain that there is (*a*) an excessive absorption of nutritive substances, both solid and liquid—a fact amply corroborated by the known etiologic factors of the disease; also (*b*) a disordered metabolism growing out of the effects of imperfect physical development, combined with too little physical exertion; (*c*) a defective elimination of waste products. That inefficient excretion through the skin, kidneys, and bowels tends to produce gout cannot be questioned, but that the disease may exist along with a normal elimination of waste products is equally certain. F. Grandmaison has carefully studied the connection between the albumin of renal disease and that of gout, and believes the association of albuminuria with gout to be a frequent one. He states, however, that during the early or premonitory stage the albuminuria is often intermittent, and hence that repeated testing is needful to avoid error.

There are a number of uric-acid theories, some of which may be briefly mentioned: 1. Garrod contends that an acute attack of gout is invariably produced by an excess of uric acid in the blood, due to increased formation and greatly decreased elimination; also, that inflammation is caused by the deposition in the joints of sodium urate. 2. Haig holds that there is a diminished alkalinity of the blood, and that the latter cannot therefore hold the uric acid in solution, so that it is deposited in the form of urates. He dissents from the view that an excess of uric acid



is formed. 3. Ebstein thinks it probable that there exists an excessive production and accumulation in the blood of uric acid. The surcharged blood excites local inflammation, followed by necrosis, and uric acid is deposited in the necrotic tissues. 4. Sir William Roberts believes that acute attacks of gout are dependent upon the precipitation of the crystalline biurate of sodium, and that an excessive absorption of urates, a defective action of the kidneys, or some other such cause induces an undue accumulation of the urates in the circulating medium. Here the urate is transformed into the less soluble biurate. 5. V. Noorden concludes that the essential process is a tissue-necrosis attributable to the presence of a hypothetic ferment, and that the uric acid, which is without etiologic effect, is deposited at the necrotic focus. 6. Klemperer<sup>1</sup> has shown as the result of observations made in cases of gout, of interstitial nephritis, etc. that as long as the function of the kidneys is not materially interfered with, the presence of considerable amounts of uric acid in the blood cannot be attributed to diminished elimination, but must be attributed to increased formation. But the presence of an equivalent of uric acid in the blood in certain affections other than gout (*e. g.* leukemia) shows that this factor is not the sole cause of gout. Again, the solvent power of the blood-serum in gouty patients has been found to be adequate for the disposal of the large amounts of uric acid present. To explain the uratic deposits we must assume that the necrotic tissues possess a greater chemical affinity for uric acid than does the blood, though the nature of the substances that excite the primary inflammation and necrotic processes is not known.

**Pathology.**—The post-mortem history of gout is concerned almost exclusively with the arthritic changes, including the uric-acid deposits. The lesions are found to vary according to the stage in which death occurs; if in the acute attack, hyperemia and swelling of the capsule, ligaments, and synovial membrane are present, together with an inflammatory exudation into the joint. As before intimated, the process begins as a localized necrosis in the articular cartilages, caused by a special but as yet unknown agent, and for these necrotic areas the urates have a strong chemical affinity, and are deposited therein. The process spreads, and may either be of uniform intensity or confined to limited areas. The deposit, which is always an interstitial one, is present from the primary paroxysm, becoming thicker as the seizures are repeated. The surface of the cartilage presents a white patch corresponding with the point most remote from the vascular supply. Finally, the entire cartilages, the ligaments, and the synovial membranes become infiltrated with masses of chalky material (tophi), which are fluid in their earliest state and contain numerous minute crystalline masses. They soon inspissate, and later become very hard and dry. In very chronic cases the affected joints may become fixed, and occasionally the skin covering the tophi ulcerates, exposing the chalk-stones. The chalky concretions have been found in various places other than in and around the joints, a common seat being the cartilages of the ears, and less frequently of the nose, eyelids, and larynx. They have always been described in the periosteum and along the tendons of the palms of the hands, where they produce a characteristic form of contraction of

<sup>1</sup> *Deutsche medicinische Wochenschrift*, 1895, No. 40, p. 653.

one or more fingers (Dupuytren's contraction). Charcot has found them in the penis.

The *kidneys* are usually involved, the changes being similar in character to those observed in the joints, and innumerable areas of necrosis, followed by uratic deposits, are seen throughout the organs, though chiefly in the papillæ. Osler says that "the presence of these uratic concretions at the apices of the pyramids is not a positive indication of gout. They are not infrequent in this country, in which gout is rare." Granular contracted kidney (chronic interstitial nephritis), with or without arterio-sclerosis, is sometimes caused by the gouty condition (*vide* Interstitial Nephritis).

The *heart* and *blood-vessels* always present changes. Gout induces arterio-sclerosis, and the latter in turn causes cardiac hypertrophy, particularly of the left ventricle. In chronic cases fatty degeneration of the heart-muscle sometimes occurs, and chronic valvulitis, with deposits of urate of soda in the valves, has been noted. Chronic bronchitis, asthma, and emphysema are among the more common changes connected with the *respiratory tract*, acute conditions being of very infrequent occurrence.

**Etiology.**—Assuming that a specific unknown cause (ferment?) exists, the factors below mentioned are to be regarded merely as predisposing.

(a) *Heredity*.—Garrod's dictum, "that more than one-half of all gouty subjects can distinctly trace their ailment to an hereditary taint," is doubtless correct, heredity from the grandparents, which is not of infrequent occurrence, being included in this estimate. If the better class of society alone be considered, the percentage will probably be still larger. It must not be forgotten, however, that patients out of pride represent other articular affections as gout. (b) *Age*.—Primary attacks are most frequent in middle life. They are rare before puberty, though exceptionally seen even in suckling infants, but after the age of puberty they become more frequent. After the fiftieth year they decrease rapidly in frequency, and are very rare in quite advanced life. It is to be recollected, however, that most cases of established gout are met with during the declining period of life. (c) *Sex*.—The arthritic form is less frequent in women than in men, while the former are disposed to the irregular type of chronic gout quite as strongly as the latter. (d) *Diet*.—Over-indulgence in the pleasures of the table, together with defective physical exercise, constitutes a potent factor, and this even in persons who are endowed with exceptional powers of digestion. (e) *Alcohol*, and particularly the fermented liquors, are among the chief favoring influences. This fact explains the relatively greater frequency of gout in certain countries (*e. g.* England and Germany), in which the heavier beers and ales are freely used, than in America, where lighter fermented drinks are more popular. (f) *Social State*.—Most cases occur among the upper class of society, but there is also a well-defined form of "poor-man's gout" due to an excessive use of malt beverages. (g) *Lead*.—Workers in lead furnish numerous typical examples of gout. Garrod found that in 30 per cent. of the hospital cases the patients had been painters or workers in lead. He also showed that the administration of lead salts to gouty persons almost invariably



determined a gouty paroxysm. Whether lead produces gout by arresting the excretory processes, especially from the bowel and kidney, and by thus inducing a fibroid change in the kidney and liver, as is held by Oliver of New Castle, is not definitely settled. Poore points out that gout produced by lead or chronic kidney trouble is constantly associated with anemia and emaciation, and forms a distinct clinical class of cases.

**Clinical History.**—1. **Acute Gout.**—The earliest manifestations of the disease are apt to take the form of a more or less typical attack of *acute arthritic gout*. The latter is usually preceded by certain prodromal symptoms, which vary in different cases, but are almost constantly similar for the paroxysms of individual cases. The patient may complain either of slight muscular cramps and articular pains, or of dyspeptic disorder, or of an asthmatic seizure; or he may exhibit mental disturbance—irritability of disposition, broken, restless sleep, and depression of spirits. In a small percentage of instances, just prior to the attack the patient feels better than ordinarily. It has been observed that immediately before and also during the early part of a paroxysm the daily amount of uric acid found in the urine is diminished, but Klemperer has shown that no relation exists between the amount of uric acid present in the urine and the character of the disease.

*The attack* generally develops in the very early morning hours. The patient awakens suffering from pains in the metatarso-phalangeal joint of the great toe, that soon become excruciating, while the joint feels as if it were tightly compressed in a vise. The local signs of inflammation—heat, redness, swelling, and excessive sensitiveness—quickly supervene. The skin pits on pressure and becomes shiny. The body-temperature rises to 102° or 103° F. (39.4° C.), and the patient manifests intense irritability.

At the end of an hour or two the sufferings abate, the fever often declines, with free perspiration, and the patient may be able to pursue his avocation. During the next day some degree of enlargement and inflammatory edema remains, and on the following night the symptoms are usually repeated in all their violence. The condition usually progresses in this manner from four to seven or eight days, though after a few days the intensity of the paroxysms is apt to lessen. After the attack the swelling subsides and there is a slight desquamation of the skin, which resumes its normal color, and the general health is often unusually good. These so-called fits of gout usually recur from time to time, the duration of the intervals depending largely upon the patient's habits or routine of life. On the whole, the first interval is apt to be the longest, while later the intermissions may not exceed two or three months. With subsequent attacks the affection is apt to spread to other articulations. There is no tendency to suppuration.

2. **Retrocedent Gout.**—This term implies the sudden transmission of the arthritic process to some internal organ. During a paroxysm the joint-inflammation may quickly disappear with an equally sudden development of intense pain in the region of the stomach, vomiting, diarrhea, faintness, and a rapid, feeble pulse. Suppressive gout may attack the heart and produce precordial pain, dyspnea, cardiac palpitation, and much anxiety of mind. It may also excite pericarditis with a fatal result. Transmission to the head, with the development of intense



cerebral symptoms (maniacal excitement, coma, and apoplexy), also occurs. Nervous phenomena, however, are more commonly due to uremic poison.

**3. Symptoms of Chronic Gout.**—Chronic gout follows the acute variety. The transition is gradual, the intervals between attacks shorter, while the attacks themselves grow milder and longer. At last the local inflammation does not appear. The condition extends to other joints: first, to the corresponding joint on the opposite side, then to the other toes and the ankles. Later, the fingers and wrists may be invaded, but almost never the largest joints (hip, shoulder). With the progress of the affection the chalk deposits slowly and gradually increase until the characteristic deformity is produced. The skin covering the tophi may ulcerate, exposing the chalk-stones. When the fingers are affected we note a deflection at the second or third joint, constituting a peculiar habitus.

Among important associated conditions are chronic gastric catarrh, arterio-sclerosis, cardiac hypertrophy with considerable functional disturbance of the heart, and "contracted kidney," forming a much complicated yet easily recognized clinical picture. If in cases of this sort the urine of a gouty person is carefully examined, and is found to contain a small percentage of albumin and tube-casts, the whole train of events becomes easy of interpretation. The cases may be divided into two classes: (a) those in which the complexion is florid and the general health vigorous; (b) those with pale, sallow facies, emaciation, and enfeeblement. These groups are chiefly dependent upon the differences in the etiologic factors.

The course of chronic gout is liable to be interrupted by acute exacerbations with fever, during which dangerous complications may arise—*e. g.* uremia, pericarditis, pleurisy, pneumonia.

**4. Irregular Gout.**—Says Sir Dyce Duckworth: "Gout manifesting itself anywhere but in a joint is to be considered irregular or incomplete." Such cases are confined chiefly to persons of gouty heritage, though I feel confident that the diathesis may be also acquired. But though the etiologic factors that produce lithemia also in time produce gout, these two conditions should be discriminated; for, while in both we usually note an excess of uric acid in the blood, in lithemia there are no tophi present, and hence no necrotic foci in the joints or elsewhere. Irregular gout, then, rarely occurs in persons who have had previous typical attacks, but should any of the conditions described below as being dependent upon the gouty diathesis be associated, or should they alternate, with acute gout, they may be properly ascribed to the latter. On the other hand, when these conditions occur in persons who are free from hereditary taint, and whose habits do not predispose them to gout, the diagnosis of irregular gout is to be made with extreme caution. It is perfectly justifiable to apply a therapeutic test when other means of diagnosis fail to clear up the given case.

The features of irregular gout are exceedingly diversified, and to describe all of them here would lead me too far, but if the preceding criteria be remembered, all forms of irregular gout can be readily recognized. The following are the more important:

(a) *Joint and Muscle Pains.*—The muscular pains may be anywhere.

and "flying" in nature, but the muscles of the back of the neck, the lumbar region, the abductors of the thigh, and the gastrocnemii are especially liable (Tyson). These pains are most severe in the early morning hours and subside as the day advances. Articular pains attended with some degree of swelling and deformity of the joints (the latter, however, not due to uratic deposits) may be of gouty origin; and, according to Paget and Garrod, Heberden's nodosities (previously described under Rheumatoid Arthritis) may present vesicular eminences due to gout.

(b) *Gastro-intestinal Disturbances*.—The symptoms referable to the intestines are identical with those presented by lithemia. In one of my cases intestinal colic followed by diarrhea put in an appearance at long intervals. Tonsillitis, pharyngitis, and even parotitis, may also be manifestations.

(c) *Cardio-vascular Symptoms*.—Just as in pure lithemia, so in atypical gout, the increased amount of uric acid usually present in the blood, by increasing the blood-tension, excites arterio-sclerosis and chronic interstitial nephritis—affections which are fully described in appropriate sections of this work.

(d) *Nervous Manifestations*.—The different varieties of headache, including migraine, are common. Sciatica and other forms of neuralgia, tingling, itching, burning sensations, and even pain in the palms of the hands and soles of the feet, are of frequent occurrence. Hot and itching eyeballs are, according to Hutchinson, among frequent manifestations; apoplexy may arise, secondary to atheroma induced by gout; and rarely meningitis (basilar) is among the gouty morbid states. The latter also include certain psychopathia—insomnia, irritability of temper, and melancholia.

(e) *Urinary Symptoms*.—The urine is highly colored, of high specific gravity, often scanty, and the standing specimen deposits lithic acid. This is not peculiar to gout, however. In many cases uric acid is in excess only at intervals, giving rise to so-called uric-acid showers, while at other times it is diminished in quantity. In this connection the results of the investigations by Klemperer are to be recollected. Gouty persons are liable to gravel: I agree with Tyson, however, in thinking that the two conditions more frequently alternate than coexist. Intermittent glycosuria is also common in gouty subjects, and may lead to true diabetes mellitus; this glycosuria may alternate with uric-acid showers. Oxaluria has been noted. Among grave secondary affections chronic interstitial nephritis, with its characteristic features (slight albuminuria and later casts), very commonly develops, sooner or later, and cystitis (with gouty hemorrhage into the bladder), urethritis, prostatitis, and orchitis, all may be dependent upon gout.

(f) *Pulmonary Disturbances*.—Chronic bronchitis, to which asthma and emphysema are frequently secondary, is often the result of podagra.

(g) *Cutaneous Eruptions*.—Eczema is frequently associated with the gouty diathesis, and I have often observed eczematous eruptions in gouty subjects, alternating with the symptoms of bronchitis or gastric catarrh.

(h) *Ocular Disorders*.—The chief eye-symptoms are conjunctivitis and keratitis (with tophi in the cornea and eyelids), iritis, hemorrhagic retinitis, and glaucoma.

**Differential Diagnosis.**—The distinction between typical acute gout and *acute articular rheumatism* is a simple matter. But when, as is rarely the case, the former manifests itself as a polyarthritis, the discrimination is sometimes difficult. W. H. Thompson has pointed out that in gouty polyarthritis, when the knees, elbows, and phalangeal finger-joints are affected, the points of greatest tenderness on transverse pressure are over the condyles. On the other hand, in acute rheumatism the cutaneous tenderness is greater, while the points of maximum tenderness correspond with the tendons anterior and posterior to the joints. Though the distribution of pain to the pressing finger may be often utilized in the discrimination of these two articular affections, in a considerable percentage of the cases reliance must be placed upon the history, especially as to antecedents, the mode of onset, the early part of the course, and the character of accompanying symptoms and complications. In a doubtful case the blood-serum may respond to the uric-acid test, as follows: Add 5–6 minims (0.399) of acetic acid to 2 drams (8.0) of blood-serum in a watch-glass; then place a linen thread in the solution and after twelve to twenty-four hours this will be incrustated with crystals of uric acid. The result is not, however, obtained exclusively from the blood of gouty subjects.

*Chronic rheumatism* is distinguished from gout by the fact that the latter disease involves chiefly the small, and chronic rheumatism chiefly the large, joints. Moreover, chronic interstitial nephritis and arterio-sclerosis, with their varied and often serious consequences, are frequently attendant upon gout, but not upon chronic rheumatism.

To differentiate chronic gout and *rheumatoid arthritis* is sometimes a hard problem, but the following table will indicate the main points of difference:

GOUT.	ARTHRITIS DEFORMANS.
Frequently hereditary.	Not so.
Causes are chiefly dietetic.	Causes chiefly nervous.
Affects males and the better classes most frequently.	Affects females and lower classes most frequently.
Begins in the big toe and extends to other toes.	Begins in the fingers, which point to the ulnar side.
Attacks are periodic.	More steadily progressive.
Deformity due to tophaceous deposits.	Deformity due to exostosis and ankylosis, and more marked.
Uric acid usually in excess.	Not so.
Renal complications and arterio-sclerosis common.	Very rare.

**Treatment.**—(1) **Prophylaxis.**—In order to prevent the development of gout, especially in persons who have inherited or acquired a strong predisposition to the disease, temperate and even rigid habits of life should be adopted. Alcohol, particularly the heavier wines (Madeira, port, sherry, champagne, etc.) and heavier malt liquors, must be eschewed, and the patient must eat sparingly of concentrated meat (particularly red meat). A residence in the country with active out-of-door exercise is of paramount importance, but straining efforts, both mental and physical, are to be avoided. The climate should be temperate and moderately dry. The sleeping apartments should be capacious, well venti-



lated, and free from draught, and the action of the skin is to be favored by cleanliness, and if the patient be strong by a cold bath in the morning with friction. For the robust, Turkish baths at intervals of two or three weeks constitute an excellent measure. In the class of patients that are pale and debilitated warm baths on retiring are preferable, and the chilling of the skin-surface is to be carefully guarded against. The patient should wear flannels next to the skin in all seasons.

(2) **Active Treatment.**—(a) *Dietetic.*—The amount of food must be lessened, the dishes being simple, nutritious, and taken at regular intervals. Fresh vegetables, containing a minimum of starch and sugar—cabbage, salads, etc.—are best, and these, together with beef (in small amounts), mutton, chicken, milk, fruits (except bananas, tomatoes, and strawberries), and stale bread, should constitute the chief dietary. Fat in the form of good butter may be taken freely—from  $2\frac{1}{2}$  to  $3\frac{1}{2}$  oz. (70.0–100.0) per diem, according to Ebstein. I have observed that occasionally patients do best on albuminoids, while, on the other hand, with about equal frequency they improve on a vegetable diet; but I am convinced that a mixed diet, such as has just been indicated, is best adapted for the vast majority of the cases. Among articles to be avoided are pastry, tea and coffee, hot bread and cakes, sweet puddings, cheese, dried meats, and all highly seasoned dishes. It may become necessary, though rarely, to administer alcohol. This is especially the case in suppressed gout, and when needed whiskey or gin (diluted) is to be preferred.

*Mineral waters*, particularly the alkaline, are highly advantageous, and sometimes are even curative. Their value, like that of the warm baths and systematic exercise, is dependent upon their power to increase renal elimination. Whether they promote solubility of the uric acid in the blood is questionable; moreover, according to the observations of Klemperer, this is not a rational indication. The carbonate and citrate of lithium are efficient diuretics, but have no other claim to virtue in this disease. Among natural waters of special value abroad are Vichy, Carlsbad, Homburg, Ems, Kissingen, Aix, Buxton, and Bath, and in this country Saratoga and Bedford. These waters are to be taken when the stomach is empty and in large quantities.

(b) *Medicinal Treatment.*—During an acute attack the pain, if excruciating, is to be relieved by a hypodermic injection of morphin, which is to be followed by a purgative dose of some mercurial. Colchicum is the specific remedy, and must be administered, in the form either of the wine or the tincture, in doses of  $\mathfrak{Mxx}$ – $\mathfrak{xxx}$  (1.333–1.999) every four hours. It alleviates the inflammation and promptly relieves the pain, but its effects during the attack should be carefully noted. After the paroxysm it should be continued, though in small doses, combined with the citrate or bicarbonate of potassium or lithium. The limb should be raised and the affected joint or joints wrapped in flannel or cotton-wool. Warm alkaline solutions or hot fomentations often afford relief in the worst cases, and anodynes may be tried locally. The diet should consist chiefly of milk, animal broths, and egg-white during the attack, while after the latter rice, eggs, fish, and other light forms of meat may be added, the more liberal dietary previously indicated being slowly resumed.

In the intervals between the acute attacks the prophylactic and dietetic measures previously mentioned are to be resorted to with a view to preventing recurrences of the disease, and in addition the alkaline diuretics and saline laxatives, together with warm bathing, will be found of the utmost value.

In chronic and irregular forms of gout medicines are of subsidiary importance, and are in no wise comparable in their beneficial effects to the previous recommendations. Two agents deserve prominent mention, however. They are piperazin and the extract of *thymus* gland. The ingestion of the latter, as obtained from the calf, is followed by an increased excretion of uric acid. Piperazin has been warmly advocated in all forms of gout for its supposed effect as a solvent of uric acid, and clinicians are almost unanimous in reporting its favorable results. Its beneficial effects are probably due to its diuretic action. The dose is gr. v-x (0.324-0.648) thrice daily, freely diluted with water. Some authors highly recommend the salicylates for acute attacks of gout, both primary and intercurrent, in the course of the chronic form. In my own experience they have been less effective in this disease than colchicum, though ammonium salicylate or salicin may be tried if there be present marked gastric disturbance, since it is better borne under these circumstances than colchicum. If nephritis or a failure of compensation be present, even the former remedies should be administered with extreme caution.

For *chronic gout* potassium iodid has been much used, though with slight advantage to the patient, I think. The bitter tonics, combined with a vegetable salt of iron, should be resorted to in the anemic, debilitated class of gouty patients, and a change of climate often serves to improve bodily vigor in the same category of cases.

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## LITHEMIA.

**Definition.**—A condition due to the faulty oxidation of nitrogenous matter. It is characterized chemically by an excess of uric acid in the blood, and clinically by various digestive, circulatory, genito-urinary, and nervous phenomena. My chief purpose in describing lithemia separately is that the common error may be avoided of attributing the symptoms of this condition to other causes.

**Pathology and Etiology.**—The deficient oxidation or the tissue-metabolism is dependent upon the same causes as in gout, but lithemia differs in being, comparatively speaking, a latent condition. The excess of uric acid may be for a time eliminated through the natural channels (kidneys, lungs, skin, etc.) without the occurrence of symptoms. On the other hand, when as the result of too little exercise, impaired elimination, high living, the use of sweet wines, combined with the neurotic temperament, uric acid is allowed to collect in different parts of the organism, marked disturbances—nervous, gastro-hepatic, etc.—follow. We are not here concerned with the articular type. Among other factors that predispose to the onset of lithemia are alcoholism, heredity, climate (temperate or cold climates favor diminished actions of the skin), and the male sex.



**Symptoms.**—The nervous, circulatory, respiratory, integumentary, and genito-urinary symptoms are practically the same as those described under Irregular Gout, but I would here emphasize the broad clinical fact that the urethral and genital mucous membranes often become inflamed on slight provocation, producing urethritis, cystitis, orchitis, epididymitis, vaginitis, endometritis. These conditions resist treatment obstinately.

**Gastro-intestinal Symptoms.**—The earliest symptom-complex is usually presented by the gastro-intestinal tract. The appetite is variable, sometimes voracious, and at other times it is impaired or perverted. The tongue is coated, particularly in the mornings, and a metallic taste is often complained of, while various forms of indigestion attend. There may be a delay in the conversion of the albuminoids, in which cases such symptoms as pyrosis, gastric oppression, fulness, and sometimes nausea and vomiting, appear soon after the ingestion of food. These symptoms, together with marked flatulence, are manifested at a later period after meals if there be failure in the digestion of the carbohydrates. The bowels work irregularly, constipation alternating with brief attacks of diarrhea, which may be attended by colicky pain, the discharges being often frothy and ill-smelling. Hemorrhoids are usual, and melena may occur, though often independently of the hemorrhoids. The liver is somewhat enlarged and often tender. A few prominent **cardio-vascular symptoms** should be mentioned, such as palpitation, which is common, appearing particularly after eating. More rarely it occurs while the patient is at rest or even lying abed, the attacks lasting from a few minutes to as many hours. Increased arterial tension develops early, but may not be constant, and is due probably to the action of the uric acid in the blood upon the vaso-motor nerves, exciting universal contraction of the arteries. This condition may be present for a long time before actual arterio-sclerosis is in evidence. The latter complaint invariably follows, however, and sooner or later the well-known group—chronic gout, arterio-sclerosis, and granular kidney—will be presented.

**Prognosis.**—This is ordinarily favorable if the condition is recognized and properly treated before irreparable changes have taken place in the arteries, joints, and kidneys.

**Treatment.**—(1) **Prophylaxis.**—The patient should be taught the lesson of thorough mastication, and robust, plethoric persons should exercise with method in the open air, with a view to consuming the fats in the body. For this purpose cycling, horseback-riding, rowing, and walking are all excellent. Nervous persons, however, demand rest (Gray). The constant use of lithia-water, more particularly in the spring of the year, is warmly advocated by Wilcox.

(2) **Diet.**—As in gout, so in the preliminary stages of lithemia, no single dietary suits all cases, though I agree with those who contend that a diet consisting chiefly of albuminoids is proper in most cases. It has been shown that a vegetable diet overtaxes the oxygenating power of the system (Porter). The lighter forms of albuminous articles of diet are to be preferred, and, if well borne, fruits and green vegetables may be added; but fried meats of all sorts and made-over dishes are to be eschewed. There are cases in which the gastric digestion is feeble, and in such the carbohydrates are better borne than the albuminoids. Cream and good



butter are the only forms of fat to be allowed. Highly-seasoned articles must be avoided, and in the use of alcohol lithemics should be guided by the same rules as gouty subjects.

(3) **Medicinal Treatment.**—If the patient be robust, it is well to begin with a saline laxative, such as Carlsbad Sprüdel salt (3j-ij—4.0-8.0), moderately diluted and taken before breakfast. If necessary, the hepatic function may be stimulated still further by a mild mercurial or by podophyllin. On the other hand, the neurasthenic, delicate sufferer must use a milder form of laxative, such as Rochelle salts in the same dose, or sodium phosphate in the morning, or a rhubarb pill at night. This class of lithemics also requires nerve-sedatives (sodium bromid, phenacetin, etc.), and diuretics to aid in the excretion of uric acid. If it be true, as some claim, that the sodium phosphate is for the greater part excreted by the urine, and that it holds in solution more uric acid than any other salt, it is one of the foremost remedies in the treatment of the affection. Personally, I have found it to be a most useful agent. To reduce acidemia and to gently stimulate hepatic activity the salts of lithium, highly diluted, may also be tried. To aid in the digestion of the albuminoids hydrochloric acid may be needful, and if the appetite be impaired it may be combined with a simple bitter or with *nux vomica* (℞x-xv—0.666-0.999) thrice daily. Nitro-hydrochloric acid should be given a trial in cases in which the hepatic symptoms are prominent.

## RACHITIS.

(*Rickets.*)

**Definition.**—A constitutional disease of childhood, exhibiting gross nutritive changes, chiefly in the bones and cartilages, causing deformities, and also in the ligaments, muscles, and other anatomic structures.

**Pathology.**—A mere summary of the anatomic characters can be given here. There is a derangement of the nutritive processes which retards and otherwise modifies the growth of the bony skeleton, particularly of the skull, the ends of the ribs, and of the long bones. The latter soften or remain unduly flexible as the result either of the absorption of ossified structures or of the greatly diminished deposition of lime-salts. Longitudinal section of the long bones shows the seat of the chief changes to be at the junction of the epiphysis with the shaft. In health we note at this point two thin layers, of which the outer (next to the epiphyseal cartilage) is the proliferative or hyperplastic zone. It presents a bluish color, and it is here that the cartilage-cells undergo division. The inner layer, exceedingly narrow and of a yellowish hue, is the so-called zone of ossification. These two narrow bands lie side by side and present perfectly regular and parallel outlines. On the other hand, in rachitis, both zones, though more particularly the proliferative, are greatly thickened, much softened, and their margins irregularly notched. The periosteum is thickened and easily separable from the shaft. A consideration of the histo-pathologic changes readily explains the gross changes just enumerated. A microscopic examination shows an increased rate of pro-

liferation of the cartilage-cells with a scanty, fibroid matrix, while the ossific layer presents disseminated and imperfectly calcified areas. Similarly, the deeper (osteoblastic) layer of the periosteum is thickened, and remains spongoid, while the cortical layer of the bones may lessen in dimensions, owing to absorption of true bone-tissue.

The above facts render easy the comprehension of the coarser changes produced by rickets. Thus we note great enlargement of the epiphyses of the long bones, obviously due to the increased activity of the proliferative layer. The cranial bones present areas of the so-called craniotabes, and yield to the pressing finger in consequence of delayed ossification or of resorption of the lime-salts in completely organized bones. The latter processes may lead to a disappearance of the cranium in certain areas, causing depressions, while flattened protuberances may develop over the antero-lateral regions as the result of augmented proliferation—changes which constitute the rachitic type of skull. When cases terminate in recovery the bones become hard and ossify, although the deformities persist. The chemist has shown us that rachitic bones may contain less than half the normal percentage of lime-salts. The liver and spleen are moderately enlarged, and rarely the mesenteric glands are increased in size.

**Etiology.**—(1) *Rachitis* may occur in the *new-born*. Schwartz states that among 500 new-born children in Vienna, 75.8 per cent. show distinct signs of rachitis. Doubtless this estimate is too high, and entirely at variance with the experience of clinicians in general; but I believe that congenital rickets is by no means a rare condition. Many of the cases are still-born, and those that outlive childhood become peculiarly dwarfed (*micromania*). (2) *Heredity*.—The instances in which rachitis develops at an early period of life, due to ante-partum causes, are not rare, but it must not be forgotten that it is extremely hard to estimate the influence of heredity where both parent and child are exposed to similar unfavorable hygienic and dietetic conditions. Ill-health, malnutrition, close confinement, lactation, and syphilis may all act as predisposing factors during pregnancy. Setting aside syphilis, and perhaps phthisis, the state of the health of the father has little if any effect in the causation of rachitis in his offspring. (3) *Geographical Distribution*.—The disease is more common by far in large cities than in rural districts, and in European countries—Russia, Germany, Great Britain, and Italy more especially—the disease prevails more extensively than in America. (4) *Race*.—The colored race furnishes a preponderance of rachitic subjects. The reason for this may be a racial need of warmth that is not supplied by the temperature of more northerly latitudes, their native habitat being in a more southerly climate. The Italian race also suffers inordinately. (5) *Station*.—It is especially among the ranks of the poor children in large cities that rachitis is seen. Joukowsky, from personal observations in over 3000 poor children in St. Petersburg examined for rachitis, found that from the working classes come the greatest number of cases. The quarters of the cities in which the poorer classes live are densely crowded, the dwellings are insufficiently ventilated, and there is a great lack of sunlight. (6) *Diet*.—The disease is dependent largely upon improper or insufficient food, and among hand-fed children the disease is much more common than among those at the breast. It also occurs in breast-fed infants when the mother's milk is poor in quality as the result of pre-



vious ill-health or too long-continued lactation. The view was at one time widely held that rickets was produced by a farinaceous diet, and that the active agent was lactic acid, produced by the fermentative processes set up by the starch. Even granting, however, that the lactic acid forms a soluble salt by union with the lime of the bone, thus removing it from the system, this does not explain the productive lesions described under Pathology. According to another view, rachitis is apt to develop when the system is deprived of an adequate amount of fat, and for this belief there is considerable experimental proof. Whilst, therefore, certain forms of diet predispose to rickets, they do so chiefly for the reason that they either are defective in certain particulars or do not supply certain necessary articles in adequate proportion. (7) *Age*.—Of 903 cases, more than 75 per cent. occurred before the end of the second year, but of these only 99 commenced during the first half year. The third year of life furnished 134 cases (Bruennische, Von Rittershain, Ritsche). (8) *Sex* is without effect. (9) *Syphilis*.—Divers views are entertained regarding the rôle played by syphilis as a cause of this disease. Doubtless the two affections are sometimes associated, and it cannot be denied that syphilis brings about a marked impairment of nutrition both in the mother and the child, so that the disease may engender a predisposition to rickets. (10) Mircoli contends for the *microbic nature* of the disease, believing that it is produced by the action of ordinary pyogenic organisms upon the osseous and nervous systems. He adduces clinical and pathological evidence in support of this position.

**Symptoms.**—The onset is slow, and the symptoms of gastro-intestinal catarrh, with their usual effect upon the general nutrition, may precede or accompany the true rachitic symptoms. At the beginning the infant is restless, irritable, and sleeps poorly, and slight fever is present in some cases. About the head and neck the child perspires freely, especially when asleep, wetting his pillow while the rest of the bed is dry. It is also annoyed by the bed-clothes, which it continually throws off, lying exposed even in a cool temperature. Among the earlier symptoms is a tenderness both over the bony surfaces and the soft parts, so that the patient wishes to keep still and dreads to be handled. The cause of this general soreness is not known, though the periosteal changes may be the main factor. The child is languid and disinclined to move his limbs or to walk or play, even if he have done so previously.

The symptoms are progressive in their development, rachitis being ordinarily a chronic disease, so that after many months more pronounced features, including various bone-deformities, appear. Owing to the impairment of nutrition of the muscles the use of the limbs may become impossible, and these cases have been spoken of by writers as “rachitic paralysis;” this, however, is a misnomer. Cases have been reported by Berg and others that resembled spastic paralysis, pseudo-hypertrophic paralysis, and other conditions of nervous origin, but their true nature is made apparent by the associated symptoms and history.

The first rachitic osteal changes are presented by the cranial bones, the ribs, the radius, and the ulna. The cranium appears enlarged, though this enlargement is more apparent than real, being due to the backward growth of the facial bones. The sutures remain open, the fontanels are large, and their closure is delayed, sometimes until the fifth or even the



eighth year. Craniotabes is most frequently seen in infants under one year of age. This soft, thin condition of the bones is due to pressure both from within and without, and is hence observed over the posterior and lateral aspects of the skull, or on the surfaces on which the head of the child rests while lying. To detect the presence of craniotabes light pressure with the fingers is to be made in a direction away from the sutures. A rachitic head generally approaches a square in outline, or it may present marked angularities, with an increase in the antero-posterior diameter and a flattened top. Hyperostoses may cause prominence of the parietal and frontal eminences, giving the forehead a square, broad outline. The veins of the scalp are enlarged, and the hairy growth is usually scanty, being often removed from the back of the head by rubbing. Drs. Whitney and Fischer first called attention to the fact that the ear placed over the anterior fontanel often detects a systolic murmur, which they regard as peculiar to rickets. A considerable patency of the anterior fontanel both in health and disease allows of detection of this murmur, however, and hence its diagnostic value is slight. A prominent feature of the disease is delayed teething, the teeth that appear being deficient in enamel, ill-shapen, and prone to early decay.

The ribs become affected very early. Anteriorly, where they join the costal cartilages, swellings occur, causing the "rachitic rosary." This is composed of nodules corresponding with the costo-chondral articulations, and these can generally be seen and always felt under the skin. They rarely outlast the fourth or fifth year. The ribs present two short curves—one at the junction of the dorsal and lateral parts of the thorax, and the other in front, where they turn sharply inward toward the sternum. This deformity is the result of the atmospheric pressure upon the softened bones, a shallow groove usually being produced in the line of the costo-chondral articulations or obliquely from the second or third rib downward and outward. These changes lessen the transverse diameter of the thorax in front and interfere with the lung-expansion in the antero-lateral portions of the chest. They also produce bulging of the sternum, resulting in the so-called pigeon or chicken breast. On both sides, from a point corresponding to the anterior end of the eighth or ninth rib, there passes outward toward the axilla a furrow (Harrison's groove) which is caused by an eversion of the lower part of the thorax, and is heightened by atmospheric pressure, particularly during inspiration. This thoracic deformity is not peculiar to rickets, but is met with in all cases in which there is moderate obstruction to the ingress of air into the lungs.

Among the first indications of rickets is an enlargement of the lower end (junction of the shaft and epiphysis) of the radius. The radius and ulna are sometimes twisted and deflected outward, owing to the fact that some of the body-weight is supported by the hands when sitting or crawling. The clavicle may be thickened and curved near either end, and occasionally the scapulæ may be enlarged, but deformities of the upper extremities are rare as compared with those of the lower. Occasionally the vertebræ and intervening cartilages soften, with a resulting spinal curvature, and in such instances there is usually an antero-posterior curvature with which lateral deflexion may be associated.

Pelvic deformities are not uncommon, and are of no little importance in female children as bearing upon the questions of marriage and subse-

quent labor. The femora may be curved, often forward and more rarely outward; swelling of the lower end of the tibia is, however, the first change to be observed in the lower extremities. In some well-advanced cases the heads of the bones forming the knee-joints are also enlarged, and outward curvature of the femora and tibia is common, especially under the age of one year (see Fig. 32). After the child begins to walk a forward bowing of these bones, due to the weight of the body and to muscular action, occurs. Knock-knee is sometimes observed. It is worthy of mention that persons who have suffered from rickets in infancy usually fall short of the average stature on reaching adolescence, rickets stunting the growth of the skeleton, and especially of the bones of the face, pelvis, and legs.

These skeletal changes sustain a causal relation to many, and some serious, affections, chiefly nervous. Thus, craniotabes is supposed to induce laryngismus stridulus, though this condition may also arise in the rachitic without cranial softening. In like manner, rickets predisposes to tetany, which affects most commonly the upper extremities. Convulsions are also prone to occur in this disease. The reflex nervous excitability is unquestionably exaggerated in rickets, and another exciting cause for the eclampsia so often met with is the gastro-intestinal catarrh that is quite generally present. The abdomen becomes greatly enlarged, chiefly by flatulence, though to a less extent also by the swelling of the liver and spleen and the contraction of the thorax. Chest complications are common. Most of them are due primarily to a mechanical interference with a cardio-pulmonary circulation, and with the function of respiration as a consequence of the thoracic deformity. Among these are atelectasis, bronchial catarrh, broncho-pneumonia, and emphysema.

**Prognosis.**—The evolution of rickets is a long process, accompanied by a slowly progressive impairment of the general nutrition; and hence most patients become weak, anemic, and emaciated. The so-called “fat rickets,” however, is not rare. Innately, the disease tends to spontaneous cure, which is attained from the end of the second to the fifth year; but its course may be abridged to a few months by appropriate treatment. When death occurs it is usually occasioned by one or other of the complications before mentioned, and especially by laryngismus stridulus or pneumonia.

**Treatment.**—**Prophylaxis.**—The institution of preventive measures is a matter of first importance, and by simple means directed to the antepartum causal factors in the mother rickets may in a large proportion of the cases be prevented. Prophylaxis also embraces appropriate feeding and other agencies that tend to maintain the normal nutrition of infants.

**Hygienic Management.**—As faulty diet is in a great measure responsible for rachitis, proper feeding is an important factor, and if the child cannot be satisfactorily nursed by its mother and if it is under the age of six months, a wet-nurse should be procured. Should this not be practicable, it must be hand-fed, and the best artificial food is cow's milk, if properly prepared. In cities it is to be sterilized, and then diluted to suit the age, and I have found that barley-water, when made in the manner recommended by J. Lewis Smith, may be added to milk, replacing the water most advantageously. A heaping teaspoonful of barley-flour is poured into 25 teaspoonsful (3iij—96.0) of water, and when the mixture is lukewarm 10 or 15 drops of diastase (Forbes) are added to it, the gruel

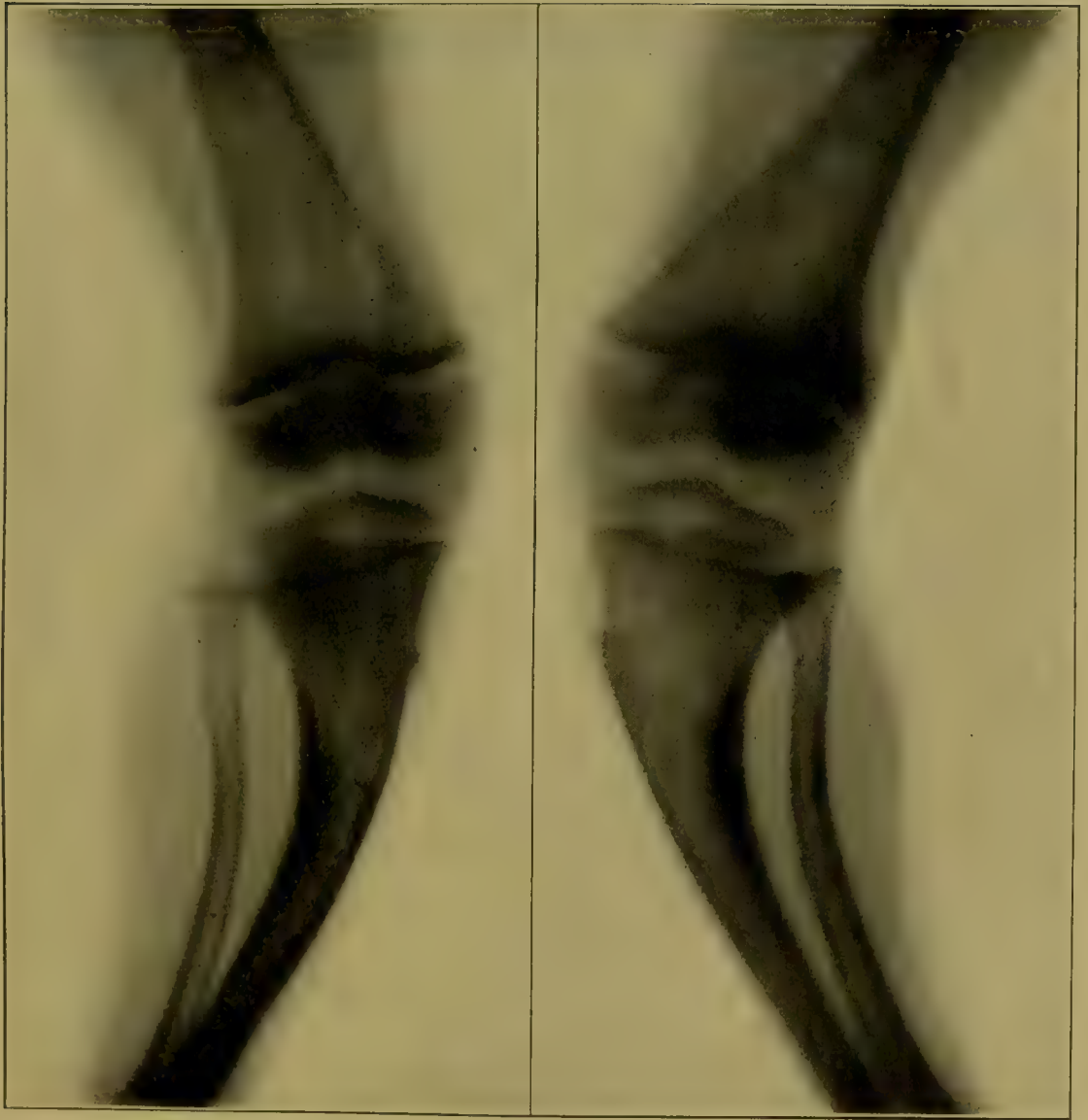


FIG. 32.—Outward curvature of tibia and fibula (Willard).





in a few minutes becoming much thinner from the digestion of the starch. The physician must regulate with much precision the frequency of the feeding, and the amount of food taken according to the age of the child. The stools are also to be inspected. If they are green or if curds appear, either digestion is imperfect or the child is being over-fed. Older children may be given the lighter meats, green vegetables, and fruits, but these must be carefully selected.

Other hygienic details are of little less importance than a proper diet. The decubitus of the child must be changed frequently, so as to prevent bony deformities; moreover, the rickety child should not be allowed to walk, and to prevent his doing so splints extending beyond the feet have been recommended. A tepid bath, warm clothing, and a prolonged daily stay in the open air are measures that should not be neglected.

Of medicines, those that rank highest are phosphorus, the hypophosphites, iron, and cod-liver oil. The officinal oleum phosphoratum (gr.  $\frac{1}{150}$ —0.0021) is used by Jacobi. Phosphorus is highly spoken of by many, and I have found it quite serviceable in my own practice. It may either be given pure (gr.  $\frac{1}{200}$  to  $\frac{1}{100}$ —0.0003 to 0.0006) or preferably in the form of an emulsion with sweet oil or cod-liver oil:

R <sub>y</sub> . Phosphori,	gr. $\frac{1}{10}$ (0.00648);
Olei olivæ,	ʒij (64.0);
M. et ft. emulsio.	

Sig. ʒj three times a day, after meals, for a child under the age of one year.

When it is desired to administer cod-liver oil and it is not tolerated by the stomach, it may be rubbed gently into the skin of the thighs and trunk. Arsenic in small doses has proved to be a capital remedy in selected cases; and iron, particularly in combination with arsenic, is indicated if anemia be pronounced.

The numerous complications to which rachitic subjects are liable present special indications which are to be met by the same measures as when they arise under other circumstances. The condition of the digestive organs must be kept constantly in mind; and no remedy, however promising, that is designed to assist the general condition should be continued if it tends to aggravate the digestive disturbance. A proper regulation of the diet will often correct the latter, but if not, suitable remedies directed to the gastro-intestinal catarrh must be employed. The treatment of the rachitic deformities belongs to the domain of the orthopedic surgeon.

## SCORBUTUS.

(*Scurvy*.)

**Definition.**—A constitutional disease, caused by a lack of fresh vegetables in the diet, and characterized by anemia, excessive weakness, spongy gums, a tendency to muco-cutaneous hemorrhages, and a brawny induration affecting chiefly the muscles of the calves and the flexor muscles of the thighs.

**Pathology.**—We know nothing concerning the pathogenesis of scurvy. Evidences of profound anemia are found upon microscopic examination of the blood, which is thin and dark, but there is no leukocytosis. The skin may show spots of subcutaneous hemorrhage (ecchymoses), but the most characteristic hemorrhage is that under the periosteum of the femora. Bleedings into the articulations and muscles may also at times be noted, and occasionally the serous membranes are the seat of hemorrhages, as well as the internal organs. Submucous hemorrhages are extremely common. The intestinal mucosa may also present ulcers. The gums are swollen, spongy, dark in color, and sometimes ulcerated, and the teeth may be loose or missing. The epiphyses, particularly of the lower end of the femora, may be congested, and rarely they are detached. The spleen is soft and swollen. The heart, liver, and kidneys sometimes show fatty and usually parenchymatous degeneration.

**Etiology.**—In former times scurvy was very prevalent among soldiers in the field and sailors at sea, and epidemics were common. Doubtless, however, it has declined in importance as a disease incident both to sea-life and to armies, but, as pointed out by Wise, it would seem that changing physiological and economical conditions may cause it to be dreaded on land as it has hitherto been on sea. According to my own observations, there certainly has been no increase in the frequency of its occurrence in Philadelphia during the last decade; but Osler states that the disease is not infrequent among Hungarians, Bohemians, and Italian miners in Pennsylvania, and I am inclined to believe that among this class of laborers the disease may have increased in frequency within recent times. It is rarely epidemic at the present day. Endemic appearances of scurvy are still common, particularly in portions of Russia (Hoffman) and elsewhere also, sweeping through prisons, barracks, almshouses, and other institutions of like kind. The majority of cases met with, however, are sporadic.

The above and many other facts point to the infectious origin of this disease. Testi and Beri have isolated a micro-organism which has been cultivated and inoculated into guinea-pigs and rabbits, producing in the latter pathologic lesions and symptoms simulating closely those of scurvy. The microbe is perfectly round and is a diplococcus. These experiments have not as yet been confirmed by other investigators.

The chief disposing factor is the long-continued use of a dietary deficient in fresh vegetables. Precisely what there is in the latter, the absence of which in the system produces scurvy, is not known to a certainty, but it is probable that it consists of the organic salts present in the fresh vegetables, which elements are requisite to normal histogenesis. Albertoni has recently shown that in scurvy of a protracted course free hydrochloric acid is absent from the gastric juice, and that the total acidity is much reduced, but this is neither so in every case nor at all stages of the disease. He found no deficiency of chlorids in the body. Peptonization is feeble.

Debilitating influences, as unhygienic surroundings, excessive muscular exercise, humidity, and cold, often play no mean rôle in causing scurvy. Mental anxiety and depression seem to have etiologic significance. The old are very susceptible, and all ages are liable to the dis-



ease. *Sex* has no special influence upon scorbutus. Starvation does not predispose to the disease.

**Symptoms.**—Scurvy has a slow onset. The earliest symptoms are generally a swelling around the eyes, over which the skin has the color of a bruise, and a pale face, which looks bloated and wears an apathetic expression. There is noticeable almost from the start a gradually increasing debility, emaciation, an inability to perform mental or physical labor, and despondency. The patient experiences arthritic and muscular rheumatoid pains and dyspnea on slight exertion.

With rare exceptions the gums swell, sometimes enormously, and become spongy, bleeding most readily. They may become ulcerated, and may be, though rarely, fungoid in appearance. The teeth often become loose, and in rare cases drop out. The breath emits an offensive odor, that is sometimes due to necrosis of the jaw. The tongue swells, though it is usually clean and often pale. In the mouth may be observed submucous hemorrhages in many cases. There is loss of appetite, but the digestion is usually good; there may, however, be constipation or diarrhea, more frequently the former. *Scorbutic dysentery* has been described by certain writers. The skin is dry and of a muddy color, blended occasionally with a greenish or greenish-yellow tinge. At the end of a week or ten days petechiæ and ecchymoses appear upon the legs, arranging themselves about the hair-follicles. These may also come out later on the trunk and upper extremities. Submucous hemorrhages may give rise to circumscribed swellings, and subperiosteal hemorrhages may occur and engender node-like protuberances. There may be frequently noticed a peculiar brawny induration, due to extensive hemorrhagic infiltration of the muscles and subcutaneous tissues, most marked in the ham and calves. The condition is not without considerable pain, particularly if the parts be touched, and in severe cases bullæ and vibices may be seen, as in a recent case of my own. Hemorrhages from the mucous channels of the body occur, and epistaxis is frequent. In bad cases hematuria, also melena and rarely hematemesis, may be observed. Blood may be effused into the serous membranes, accompanied sometimes by inflammatory changes in the latter; also into the lungs, which are rarely the seat of secondary pneumonia. Pulmonary infarction occurs, but is a rare event. Hemoptysis may be a symptom of the lung-complications or may occur as an independent phenomenon.

The *heart* may present symptoms, such as palpitations, feeble impulse, arrhythmia, and sometimes a basic blood-murmur, but these are without diagnostic importance. The pulse is soft, small, and on exertion much accelerated. The temperature is sometimes subnormal, and the presence of fever is a certain indication of the existence of some complication.

The *nervous symptoms*, aside from the profound mental depression, are not prominent. Insomnia may be a distressing symptom. Delirium (late) is sometimes witnessed. Meningeal hemorrhage may supervene. Both night-blindness and day-blindness are among the rarer and extraordinary ocular features.

The *urinary symptoms* vary in different cases. Albuminuria is, however, common. The specific gravity of the urine is increased, the color high, and the solid constituents diminished, except the phosphates, which are abundant. Albertoni found the proportion of chlorids less than the

normal, while other investigators claim that the percentage is high. The bones in long-standing cases may be intensely congested and sometimes necrotic, and the epiphyses may separate from the shafts. In one of my own cases an old cicatrix reopened.

**Diagnosis.**—This rests upon the following points: the history, the peculiar facies, the spongy and swollen gums, the gingival and cutaneous hemorrhages, the progressive loss of strength and energy, great mental depression, and the speedy recovery after an appropriate regimen. Scurvy will be distinguished from *purpura* under the description of the latter disease.

**Prognosis.**—Unless far advanced, the prognosis generally becomes good upon the institution of correct dietetic principles. If the disease have made extensive inroads, the danger to life is considerable. The gravity of the internal symptoms (particularly pulmonary) is far greater than of the external, and, indeed, the presence of the latter is a favorable omen. Certain complications augur a serious termination, such as pneumonia, hemorrhagic infarctions of the lung, pleurisy with bloody effusion, dysentery, acute nephritis, etc.

**Treatment.**—**Prophylaxis.**—By carrying out the known means of prevention the disease has been diminished more than 90 per cent. among mariners and soldiers. This change has been brought about by the enforcement of governmental regulations which demand that an adequate supply of antiscorbutic articles of food must be provided for military campaigns and for long sea-voyages. Fresh fruits and vegetables can be readily transported in hermetically sealed jars or cans.

**Treatment of the Attack.**—The chief indication is to be met by the use of fruits and fresh vegetables. Of the former, two or three lemons daily or oranges and other fruits suffice to work a surprising degree of improvement in a short space of time. Antiscorbutic vegetables (potatoes, water-cresses, raw cabbage, lettuce, saur-kraut) in liberal quantity should also be given. Meats, eggs, milk, and farinaceous dishes are not to be prohibited, since the patients require all forms of food to invigorate the system and to render normal the constitution of the blood; but if the digestive power be feeble, it is advisable to begin with the juice of oranges or lemons, conjoined with meat-juice, egg-white, milk, and light farinaceous articles, adding the stronger forms of animal food and fresh vegetables when improvement is noted. We may assist the digestive function by the use of simple bitters, strychnin, and hydrochloric acid (after meals), but these remedies are not needed in the majority of instances.

*Special symptoms* may call for appropriate measures. Constipation requires simply an enema. On the other hand, diarrhea presents an indication for intestinal antiseptic and astringent remedies. The oral condition varies, hence the measures to relieve it vary also; but if ulcers be present, the solution of potassium chlorate is best. For swelling of the gums the application by means of a cotton swab of tannic acid (2 per cent.) or a solution of silver nitrate (2–5 per cent.) is serviceable. A combination of boric and carbolic acids in a solution of suitable strength may be used as a mouth-wash. If copious hemorrhages occur, hemostatics are eminently useful. The various complications must be met by the usual measures, according to their nature.



## INFANTILE SCORBUTUS.

**Definition.**—A constitutional disease, characterized by the same symptoms as scurvy in adults, except that in many instances undoubted evidences of rachitis are associated.

**Pathology.**—The bones are thickened and excessively sensitive, owing to a marked subperiosteal hemorrhage, with more or less maceration, and want of firmness between the epiphysis and shaft. The muscles may also be the seat of effusion. The characteristic lesions of rickets are often associated.

The nature of the affection may be regarded as being somewhat obscure. Originally looked upon by most observers as acute rickets, it was subsequently described by Cheadle (from the clinical side) and Barlow (from the anatomo-pathologic side) as infantile scurvy. On the other hand, Ashby of Manchester, Fürst and other German writers, are inclined to the view that the affection should be considered a hemorrhagic form of rachitis. Northrup has reported 36 cases. The belief that rickets predisposes to scurvy, but that the two diseases are not regularly combined, is probably the correct one. Rachitis and scorbutus both occur in children of about the same age, and their causes are in some respects the same. Thus the dietetic factor is the chief in both diseases, but disproving the identity of rickets with scurvy is the fact that the former does not develop unless the diet fails to supply some elements contained in fruits and fresh vegetables.

**Etiology.**—Scurvy is almost solely confined to hand-fed infants, especially those reared upon the numerous infant foods which have been foisted upon the market, including condensed milk, etc. Louis Starr, Jacobi, and others have shown conclusively that it sometimes follows the prolonged use of sterilized milk, either exclusively or in combination with artificially prepared foods. It develops usually between the sixth and eighteenth months of age.

**Symptoms.**—The skin presents the muddy color peculiar to the disease in adults. The patient may be well nursed, but more often there is a tendency to wasting, and other symptoms of impaired nutrition appear, particularly irritability and disinclination to exertion. The more characteristic features appear after one or two months, and the child cries when handled, especially on touching the lower limbs. About the same time there is an irregularly cylindrical swelling of one of the thighs, due to subperiosteal effusion. Soon the other limb is similarly involved, though not always to a like degree. At first the legs are flexed, but later they become straightened and slightly everted on account of the progressive hemorrhage or separation of the epiphyses. The bones in other portions of the body may be involved secondarily in more or less rapid succession, but the swellings are less marked than in the lower limbs. Later, if teeth be present, the gums may swell and become spongy. Ecchymoses in the form of petechiæ appear upon the skin-surface, and particularly about the eyes. Barlow describes a remarkable ocular phenomenon: "There develops a rather sudden swelling of one eyebrow, with puffiness and very slight staining of the upper lid. Within a day or two the other lid presents similar appearances, though they may be of less severity. The ocular conjunctivæ may show a little ecchymosis or may be quite free."



Hemorrhages from the mucous surfaces may finally put in an appearance. To complete the statement of characteristic features, it should be mentioned that rapid improvement invariably follows an antiscorbutic regimen.

**Diagnosis.**—To distinguish *rickets* from infantile scurvy Barlow's brief though clear aggregation of the characteristics of the latter disease may be quoted: "(1) Predominance of lower-limb affection, in which there is immobility going on to pseudo-paralysis; excessive tenderness; general swelling of the lower limbs; skin shiny and tense, but seldom pitting, and not characterized by undue local heat; on subsidence revealing a deep thickening of the shafts, also liability to fracture near the epiphysis. (2) Swelling of the gums about erupted teeth only, varying from definite sponginess to a minute, transient ecchymosis."

**Prognosis.**—Favorable, even in well-established instances, if brought under the proper regimen.

**Treatment.**—An antiscorbutic dietary—fresh milk, meat-juice, and orange- or lemon-juice—meets the main indication. If there be systemic exhaustion—a condition that is not infrequent—gentle stimulation with brandy (highly diluted) and an abundance of fresh air are pre-eminent among the measures to be employed. Iron, arsenic, and cod-liver oil may be needful to complete the cure, but usually the simple means already mentioned will prove effective. The limbs, especially the lower, may claim attention. Local treatment, however, is rarely necessary, except there be separation of the epiphyses, when suitable splints are to be applied.

## PURPURA.

Two main groups are to be distinguished: (1) **Secondary purpura**, which occurs from a great variety of causes and in numerous affections, in which its clinical significance has been pointed out in appropriate sections of this work. It seems pertinent, however, to enumerate the chief among the diseases and conditions under which it may arise, as follows: (*a*) scurvy; (*b*) acute, infectious diseases (cerebro-spinal meningitis, variola, measles, septicemia, ulcerative endocarditis); (*c*) hemophilia; (*d*) numerous chronic affections, as nephritis, leukemia, pernicious anemia, jaundice, Hodgkin's disease, and tuberculosis; (*e*) malignant sarcomata; (*f*) nervous affections, as locomotor ataxia, acute and transverse myelitis, and hysteria; (*g*) mechanical causes, straining efforts, intense paroxysms of whooping-cough, and violent convulsions; (*h*) certain drugs may produce a petechial eruption—quinin, copaiba, belladonna, ergot, mercury, and the iodids; (*i*) snake-poisons produce rapid and extensive hemorrhagic extravasations, as shown by the careful studies of S. Weir Mitchell.

(2) **Primary or idiopathic purpura** forms the second group. It is divisible into (*a*) simple purpura (*purpura simplex*); (*b*) arthritic purpura, of which two varieties may be recognized: (1) *peliosis rheumatica*, and (2) *Henoch's purpura*; (*c*) hemorrhagic purpura (*purpura hæmorrhagica*).

(*a*) **Simple Purpura.**—The cause is unknown. Among predisposing

influences, however, is *age*, the condition being most common in children about the time of puberty. It may be a sequel of the acute, infectious diseases, and in not a few cases develops in seemingly healthy subjects.

*Symptoms.*—This is the mildest variety of primary purpura. The hemorrhages into the skin take the form of petechiæ, vibices, or ecchymoses. The first are extravasations of blood in the form of minute points, that appear, as a rule, in the hair-follicles, and, unlike the erythemas, do not disappear upon pressure. The vibices receive their name from the fact that the hemorrhages occur as streaks, while the ecchymoses are larger, but similar in nature and behavior to the petechiæ. They may exceed in size that of a split pea, and their hue ranges from a deep red to a livid bluish tint. As they fade away they assume at first a yellowish-brown, then a yellow color, and finally disappear. The eruption appears in a series of crops, and its seat of election, often favored by the erect posture, is the legs. Bloody serum may be effused into bullæ or large blebs. Shepherd and others have reported cases in which the purpuric eruption ended in gangrene, though in Shepherd's case the gangrene was believed to be due to the use of sodium salicylate.

(b) **Arthritic Purpura.**—(1) *Peliosis Rheumatica* (*Schönlein's Disease*).—The cause of this remarkable disease is unknown. Formerly many writers inclined to the view that it is of rheumatic origin, and since endocarditis and pericarditis are occasionally observed in association with peliosis rheumatica, considerable coloring is given to this belief. On the other hand, the fact that the cardiac complications are rare in arthritic purpura shows that not all cases of the latter disease are genuinely rheumatic. It occurs chiefly in males from the twentieth to the thirtieth year of age. Among the prodromata are angina, slight articular pains, headache, loss of appetite, and fever ranging from 100° to 102° F. (37.7°–38.8° C.). The affection is especially characterized, however, by polyarthritis, the joints being swollen, painful, and very tender; also by purpura, associated or not with urticarial wheals or erythema exudativum; and by subcutaneous edema. The purpuric eruption is the only symptom that has pathognomonic significance, and in this affection it shows a strong preference, as regards distribution, for the affected joints and the legs. The eruption, as already intimated, does not display constant characteristics. It may not differ from that of simple purpura, and the rash consists of petechiæ, ecchymoses, streaks, and rarely of bullæ (*pemphigoid purpura*); or it may be made up of wheals of urticaria, attended with intense itching; and, finally, it may be identical with erythema nodosum. These forms of eruptions may be variously combined.

*Hemorrhages* from the mucous surfaces rarely occur, though epistaxis is the most common. The extent of the *edema* varies greatly, in rare cases being quite extensive and overshadowing all other symptoms (febrile purpuric edema). *Albuminuria* may be noted, and accompanying the purpuric eruption there will be a mild febrile movement. Convalescence is usually protracted (even into years), and is often interrupted by recurrence of the characteristic features.

The *diagnosis* is made from the presence of three characteristic symptoms—polyarthritis, a purpuric rash, and edema. The combination of purpura and urticaria is one of the chief distinguishing features. It is not always possible to eliminate *rheumatism*, but the non-rheumatic cha-



racter of some of the cases may be clearly shown by the therapeutic test, as happened in one of my own patients.

*Prognosis.*—This type of the disease is generally benign, death being very rare. Complications, however, may prove serious, especially the cardiac. The throat-condition may outlast the attack, and terminate in gangrene of the uvula or tonsils.

(2) *Henoch's Purpura*—Henoch and Couty have described a form of rheumatic purpura occurring chiefly in children, and characterized by painful and sometimes swollen joints; by a purpuric eruption, plus erythema multiforme; by vomiting, diarrhea, and intestinal pain; by localized edema of the skin; and by hemorrhages from the mucous membranes and sometimes into the kidneys.

The *diagnosis* is difficult in proportion to the scanty development of the purpuric symptoms, some of which are often wanting.

The *prognosis* is favorable, though complications of more or less serious import may arise. One of Osler's cases proved fatal with the symptoms of acute hemorrhagic Bright's disease.

(c) *Purpura Hæmorrhagica (Morbus Werlhofii)*.—This is the severest form of purpura, and its apparent etiologic connection with certain infectious diseases, particularly rheumatism, malaria, etc., is interesting, but not well understood. The disease is perhaps most common in young females, particularly if they have fallen into general ill-health; but all persons are liable, and post-mortem anatomo-pathologic pictures of the disease leave little room for doubt that it is an infectious complaint.

*Symptoms.*—Prodromal symptoms, such as malaise, headache, depression, and anorexia, generally appear, and last one or two days. The invasion is moderately abrupt, with fever, and soon cutaneous ecchymoses appear upon the skin, quickly growing larger in size as well as multiplying in numbers. Slight hemorrhages from the mucous membranes into the internal organs occur. Epistaxis generally comes first; it tends to persist and to recur frequently, and the same peculiarities pertain to bleedings from other points. Prostration now becomes rather marked, the patient complaining of pains in the limbs, loins, abdomen, and chest, and the latter often presage a fresh hemorrhage. There is moderate fever, as a rule, the temperature during the height of the attack ranging from 101° to 103° F. (38.3°–39.4° C.), or it may reach 104° to 105° F. (40.5° C.), though rarely. The pulse is accelerated (120 to 130 per minute), but full and regular, though in the worst cases it becomes small and very rapid. The mind is usually clear. Hematuria occurs when hemorrhage has taken place into the kidneys, and may lead to nephritis.

There is *anemia* varying in intensity with the extent of the hemorrhage and the severity of the type, and showing the characteristics of symptomatic anemia. The face may be exceedingly pale and anxious. The course is run in from seven to ten days in mild cases, while the severer attacks pursue a longer course. It is to be recollected, however, that the type of the disease may be malignant (*purpura fulminans*) and arrive at a speedy fatal termination.

The *diagnosis* of purpura hæmorrhagica rarely presents any difficulty. Scurvy may simulate it in some particulars, but is distinguished by its chief etiologic factor—a diet deficient in fresh vegetables and fruits—by the spongy, swollen condition of the gums, the loosened teeth, and brawny



induration of the limbs. Moreover, in purpura hæmorrhagica the hair-follicles do not occupy the centers of the ecchymotic spots, and the hemorrhages from the mucous membranes are more copious than in scurvy. Malignant types of the eruptive fevers distinguish themselves by the history of the prevailing epidemic, by the characteristic prodromes and invasion, and by the high temperature. It must be remembered, however, that variola purpura often pursues an afebrile course.

**Prognosis.**—Grave, except in mild cases. In the malignant type death may come before hemorrhages from the mucosa appear. Certain complications may prove fatal—cerebral hemorrhage, inundation of the lungs with blood, Bright's disease, and shock from rapid, profuse bleedings. Death may also be the result of exhaustion due to protracted bleedings.

**Treatment.**—(a) The management of *secondary purpura* is embraced, in other portions of this volume, in connection with the treatment of the diseases and conditions which it accompanies.

(b) *Simple purpura* demands arsenic, first in moderate doses, and then increased until slight toxic effects are noticeable. Legroux speaks in warm terms of the iron compounds, and especially of iron perchlorid in doses of ʒss-j (2.0–4.0) daily, and if the child is somewhat anemic, the inhalation of oxygen will promote hematosis. The disease also requires fresh air in abundance and a generous diet.

(c) *In peliosis rheumatica*, in addition to the measures recommended in purpura simplex, the salicylates may in some cases influence the affection of the joints.

(d) *Purpura Hæmorrhagica.*—I have found that an abundance of nourishment, by supporting the patient's power, is of the greatest service. Internally, ergot, turpentine, tincture of the chlorid of iron, acetate of lead, and dilute sulphuric acid enjoy the widest reputation. The following combination, recommended by Hardaway, I have found to be very useful:

R̄. Ext. ergotæ fl.,  
Tr. ferri chlorid.,                      āā. fʒij (64.0).—M.  
Sig. Three to ten drops in water, t. i. d.

## HEMOPHILIA.

(*Bleeder's Disease.*)

**Definition.**—An hereditary affection, transmitted by females who are themselves not affected (Nasse's law). It is characterized by frequent uncontrollable hemorrhages that are either spontaneous or due to slight traumatism.

**Pathology.**—The constitutional changes or peculiarities on which the disease depends are to be found in the blood-vessels rather than in the blood itself (Henry); microscopic changes have been found in the arterioles, the middle muscular tunic being either absent or much atrophied. Vaso-motor influences also play an important part in causing an attack, as is shown by the frequent flushings of the face preceding an

attack, and also by the fact that bleeding may follow emotional excitement (Henry). Synovitis with hemorrhages into the joints may sometimes be observed. The blood presents no appreciable change.

**Etiology.**—Hemophilia is more distinctly hereditary than any other known disease, but Nasse's law is not of such universal application as is generally supposed. R. Kolster found that of 50 hemophilic families, 18 cases followed this law, 16 others with some exceptions to its provisions, and 12 without any regard to it. The law embraces the following points: The daughter (not herself affected) of a bleeder transmits the tendency to her sons, who become bleeders; her daughters do not suffer, but in turn transmit the disease to their sons. Females, however, may be bleeders, and, according to Virchow, one woman is affected to every seven men. The disease has been traced for centuries in a few families.

The disposition may be acquired, but of the conditions that may lead to its development we are entirely ignorant. It is observed in all classes of society, and is most frequent in families whose members are large, vigorous, and have delicate complexions, the complaint usually manifesting itself before the end of the second year of life, though exceptionally as late as puberty.

**Symptoms.**—The occurrence of profuse and persistent bleedings that are either spontaneous or the result of slight injury characterizes hemophilia. The character of the injuries that lead to dangerous bleedings is often exceedingly trivial; thus a slight scratch, cut, blow, the extraction of a tooth, and other minor surgical operations (*e. g.* circumcision) may be followed by severe hemorrhage.

If we include spontaneous hemorrhages, bleedings take place most frequently from the nose. Legg has made three clinical groups, based on the intensity of the symptoms, as follows: (1) Seen most frequently in men, and characterized by external and internal bleedings of all kinds and by joint-affections; (2) most frequent in women, and distinguished by spontaneous hemorrhages from mucous membranes only; and (3) characterized simply by ecchymoses.

The capillaries ooze blood—a process that may vary in duration from a few hours to as many weeks. A fatal result may thus occur in a few hours, while, on the other hand, recovery may follow a slow oozing of blood that has continued for many days. In the latter instances profound anemia follows, the blood, however, being rapidly replaced. Extensive blood-extravasations (hematomata) usually follow contusions. Petechiæ, when they occur, are apt to be spontaneous. The blood coagulates, except in long-standing hemorrhages, when it becomes thin and watery (late).

*Arthritic symptoms* are common, the larger joints, and especially the knees, being most frequently affected and showing swelling that is due chiefly to hemorrhages into the joints. In other instances febrile synovitis may be present, resembling rheumatism. The joint-symptoms may either announce an approaching hemorrhage or pain alone may be experienced. The attacks are liable to recur, especially in cold, damp weather, and may result in stiffened, deformed joints (Musser).

**Diagnosis.**—When persistent capillary oozing occurs in a person with a clear, hereditary disposition the diagnosis is clear. Without an inherited tendency we cannot be certain of the diagnosis unless pro-

tracted hemorrhages from insufficient causes are repeatedly manifested. The presence of joint-involvement is very helpful.

**Differential Diagnosis.**—*Peliosis rheumatica* is an affection which, as Osler remarks, touches hemophilia very closely, particularly in the relation of the joint-swelling. It is true that the former may also show itself in several members of a family, but the presence in this affection of more or less edema, and often of wheals of urticaria, accompanied by intense itching, aids greatly in its elimination.

**Prognosis.**—In undeveloped forms the outlook is not particularly grave, since in these the tendency may either lessen or become altogether arrested after childhood. In the majority of well-marked cases the children do not survive this period. On the other hand, those who live to become full-grown show a diminished, and in a small class of cases an absolute, disappearance of the tendency. The first hemorrhage rarely proves fatal. Boys suffer from a more serious form than girls. Moreover, menstruation, though sometimes very copious, does not to any great extent endanger the life of a hemophilic woman. Of 130 cases of pregnancy and labor, the death of the mother occurred in only 3, and abortion in 16 cases (Kolster).

**Treatment.**—The physician can do most in the direction of prophylaxis. All surgical operations that are not absolutely necessary must be avoided; neither should the teeth be erupted nor the operation of circumcision be permitted, nor are leeches permissible. Females who belong to bleeder families, as well as males who have had hemophilia, should not marry.

During the attack absolute rest—mental and bodily—must be enjoined, and light compression, and if this fail strong pressure or styp-tics, should be tried. It is, however, a great question how far agents that destroy the already weakened tissue are useful. In epistaxis ice, tannin, and turpentine should be tried before using nasal plugs; and if the latter prove indispensable the lightest only should be employed. J. Greig Smith regards lint saturated with spirits of turpentine as the best local application in epistaxis. Internal medicines are of secondary importance, though they may be tried, and opium is unquestionably of signal value, since it tends to quiet the patient, thus favoring repose. The remedies that have been given are various. Delafield, Fürth, and others have used successfully the fluid extract of *hydrastis canadensis*, the dose being from 20 to 40 drops daily; among other hemostatics, gallic acid, turpentine, and iron perchlorid produce the best results. The dose of the latter should be 3ss (2.0) every two hours, with a purge of sulphate of soda (Legg). During convalescence arsenic, iron, and the bitter tonics, together with a liberal dietary, will aid recovery.



## HEMORRHAGIC DISEASES OF THE NEW-BORN.

(a) **Epidemic Hemoglobinuria** (*Winckel's Disease*).—This affection, which is septic in nature, is occasionally met with in lying-in hospitals, and occurs in children from one to ten days after birth. The infants refuse the breast and show hematogenous icterus; gastro-enteric catarrh is an attendant of the disease. The stools are meconic; the urine is scanty, dark-colored, often albuminous, and may contain casts and epithelium. Hemorrhages occur into organs other than the kidney and into the mucous membranes, there also being mild fever, rapid emaciation, and often mild convulsions. Recovery may take place, though it is a very fatal disease. Bacteriological experiments have shown that the disease may be produced by the growth of the colon bacillus in the buccal epithelium of infants, but these investigations need confirmation before we accept their results as conclusive.

(b) **Acute Fatty Degeneration of the New-born** (*Buhl's Disease*).—This disease may be similar to Winckel's in nature. It was first described by Hecker and Buhl as an infectious disease of the new-born, characterized by cyanosis, jaundice, and copious visceral hemorrhages. The chief pathologic change is an acute fatty degeneration of the internal organs.

(c) **Syphilis Hæmorrhagica Neonatorum**.—Either at birth or soon thereafter bleedings take place into the skin (ecchymoses) and from the mucous surfaces and the navel. Jaundice may be associated. The viscera are found upon post-mortem examination to be the seat of syphilitic lesions.

(d) **Morbus Maculosus Neonatorum**.—Hemorrhage from the gastrointestinal mucosa of the new-born (*melæna neonatorum*) occurs, and may be due to intracranial lesions during birth; it may also take place independently of the latter. Preuschen has collected the reports of 37 cases, in 5 of which the brain was examined, and all of these showed cerebral hemorrhages. The latter may occur in spontaneous births and give rise to *melæna neonatorum*. Gärtner believes the disease to be an infectious one, and claims that in 2 cases he was able to identify a bacillus for which the navel is believed to be the entrance-point. The blood may also come from the mouth, nose, navel, etc. Townsend found morbus maculosus neonatorum in 45 cases in 6700 deliveries, and in most of these instances the bleeding was general. The hemorrhage usually sets in during the first week, rarely later, and the duration of the disease is between one and seven days, the mortality being a little over 50 per cent. Vomiting of the blood which the child has drawn from the breast must not be confounded with true *melæna*. The treatment is by gallic acid and ergotin, the latter hypodermically; stimulants may also be required, and warmth to the extremities if the peripheral circulation be feeble.

## PART III.

# DISEASES OF THE BLOOD AND THE DUCTLESS GLANDS.

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### ANEMIA.

**Definition.**—A pathologic condition, characterized either by a diminution in the quantity of blood or by a deficiency in one or more of its constituents. Anemias may be subdivided into—I. Primary or Essential (simple, chlorotic, and pernicious); II. Secondary (symptomatic); III. Leukocytosis; IV. Leukocythemia (splenic, myelogenic, and lymphatic).

**Pathology.**—Anemia, in its different forms, is characteristic of diseases of the blood or of the blood-making organs. It may be manifest, on examination, as a diminution of the total quantity or body of the blood (*oligemia*); of the number of red corpuscles (*oligocythemia*); of the hemoglobin (*oligochromemia*); and of other constituents, as albumin (*anhydremia*). The diminution of hemoglobin gives rise to the most obvious sign of anemia or impoverished blood—namely, the pallor of the cutaneous surface—but it is important to point out here that the quantity of hemoglobin in the blood is not necessarily proportionate to the number of red corpuscles. Thus the percentage of hemoglobin contained by the red corpuscles may vary in disease, so that a reduction in its amount does not necessarily involve a corresponding decrease in the number of red corpuscles. Conversely, a diminution in the number of the latter may not be accompanied by a proportionate diminution in the amount of hemoglobin, the corpuscular richness in coloring-matter being quite normal. As a matter of fact it frequently happens that oligochromemia is associated with a certain degree of oligocythemia, and *vice versa*, though where they coexist the degrees of reduction may neither be relatively nor proportionately equal.

Anemia can be positively ascertained only by an adequate examination of the blood. It may be inferred from the presence of pallor, languor, dyspnea, palpitation, etc.; but it should be borne in mind that not every pale person has anemia, since pallor of the face may be hereditary, and, at the same time, perfectly consistent with good health, a normal number of corpuscles, and a normal percentage of hemoglobin. Conversely, a person with marked vascularity of the face, and a rosy complexion even, may have anemia.

The anemias embrace those conditions, also, in which there are changes in the shape of the red corpuscles (*poikilocytosis*), and in their size (*micro-, macro-, or megalocytosis*).

## I. THE PRIMARY OR ESSENTIAL ANEMIAS.

Primary anemias constitute those forms in which, so far as our present knowledge of their etiology and pathology goes, no other tissues or organs than the blood and the blood-making organs are either at fault or are directly affected. Future investigations of the life-history of the blood may reveal the exact causation of what are now regarded as primary or essential anemias, and thus permit of a clearer discrimination and a more accurate classification.

## SIMPLE OR BENIGN ANEMIA.

This form is not infrequently met with as a congenital, constitutional affection, without any assignable cause, and is entirely free from pernicious manifestations or tendencies. There is no discoverable element of relationship between simple benign anemia and chlorosis, nor is the former symptomatic of any disease in which anemia is common, such as tuberculosis, carcinoma, and nephritis.

**Etiology.**—Simple constitutional anemia is often met with among the poorer classes, and from this fact it is probable that living or working in a vitiated atmosphere, as well as deficient sunlight and nutriment, is primarily active in reducing the general health. In this way is often caused a lifelong pallor, due to an interference with the normal process of blood-making (*hemogenesis*). There are also certain individuals in whom slight pallor and systemic feebleness have existed from infancy (thus probably congenital), and whose modes of life and environment have been more or less uniformly hygienic and provident. In such cases we may assume that there is some innate imperfection—*anatomic or physiologic*, or both—in the blood-forming organs.

Finally, in the later manifestations of slight general anemia a developmental strain or abnormality may start a disorder of hematopoiesis in organs congenitally insufficient for new and greater demands for blood made by the system.

**Symptoms.**—There is some pallor, often with languor, slight palpitation, and dyspnea, occasional headache, and a tendency to fatigue. The general health is not otherwise disturbed, and an active life may be enjoyed for many years. Examination of the blood shows a slight reduction in the number of the red cells and of the hemoglobin (relative). This degree of anemia persists without aggravation or amelioration. It may be found to affect males and females, and is observed principally in adult life.

The **diagnosis** of simple, benign, or constitutional anemia should be made with considerable caution and reserve, and it should be arrived at only after the closest scrutiny of all the symptoms and signs; the most careful study and judicious balancing of the data entering into the previous history of the patient. If there be a latent or incipient tuberculosis, carcinoma, or nephritis, a previous attack of some infectious fever, rheumatism, etc., this fact clearly bears upon the case, and the diagnosis of simple anemia is precluded.

The **prognosis** is usually favorable. On account of the possible existence of one of the above-mentioned diseases, or from the fact that



a grave variety of anemia may be superadded, however, it should be guarded in the mind of the physician, at least.

The **treatment** of simple, benign anemia is an expectant one in most instances. Hematinics (iron, arsenic, etc.) are seldom required, as they have little if any influence upon the blood or upon the pallor or other symptoms. A rigid system of hygiene, together with attention to proper food and drink and to the manner of eating and drinking, will probably ensure to the patient all the benefit that may be obtained. Cardiac tonics (digitalis, strophanthus) may be useful in controlling the palpitation. It is worse than futile to attempt to eradicate any congenital defect of the blood-vessel system or hematopoietic organs.

#### CHLOROSIS.

(*Green Sickness.*)

**Definition.**—A blood-disease, occurring chiefly in adolescent females, and characterized principally by a deficiency of hemoglobin in the red corpuscles. It runs a mild course, though with a tendency to relapse.

**Pathology.**—It is so seldom that death occurs in cases of chlorosis that autopsies of this disease have not been frequent enough to determine definitely the nature of the findings. There is no loss of fat in the body, but signs of physical degeneration and disorders of development are quite common, hypoplasia of the vascular system and of the genital organs seeming to be the most prominent. Incurable cases of chlorosis are nearly always characterized by anomalies of the blood-vessels and genitalia (Rokitansky). Virchow has also shown that congenital arrest of development of the aorta and larger arteries, as indicated by their small size, their soft and elastic walls, is quite constant in chlorotics. The uterus (especially) and adnexa manifest the hypoplasia, and yellowish spots and streaks of fatty degeneration are sometimes seen in the intima of the arteries. The cardiac muscle is softened, the whole heart is dilated, and the left ventricle is usually somewhat hypertrophied.

**Etiology.**—Chlorosis occurs most frequently in girls at or near puberty, and also may appear between that period and twenty or twenty-five years of age. It usually happens that the condition dates from a scanty menstruation, beginning late in the “teens,” but it should be recollected that amenorrhea is not, as formerly supposed, a cause, being rather an effect of the underlying blood-disorder. Blondes are oftener affected than brunettes. In males the disease is rare, though cases may develop at puberty or during adolescence.

The influence of *heredity* in the causation of chlorosis is undoubted in those cases described by Virchow, in which congenital hypoplasia of the blood-vessels and genitalia is found to exist. Other cases, moreover, in which such anatomic evidence is wanting, also bear the stamp of heredity, in that their mothers have been, and their sisters are, chlorotic. A *family tuberculous taint* may predispose to chlorosis (Jolly). Such *unhygienic conditions* as bad air, dimly-lighted rooms, a lack of nutritious food and out-door exercise, a sedentary occupation, hasty and irregular eating, excessive tea- and coffee-drinking, irregular and insufficient hours of rest and sleep; bodily fatigue, as from stair-climbing and standing in

constrained positions without intervals of rest,—all these predispose to the disease. And yet girls living amid the most luxurious and favorable surroundings have had chlorosis. It is not at all improbable that, as the late Sir Andrew Clarke believed, *copremia*—the absorption of the toxic ptomains and leucomains from the colon in constipation—is often the cause of chlorotic symptoms and signs, though physiological chemists fail to find in the urine the evidences of intestinal putrefaction (*i. e.* an increase of the aromatic sulphates). Sometimes a previously existing simple constitutional anemia appears to be an underlying cause for an exacerbation of genuine chlorosis. In such instances, however, I believe additional exciting causes to be operative.

Sudden emotional excitement and prolonged mental over-exertion operate as causative agencies. Shock from bad news, such as loss of relatives, crosses of various kinds, home-sickness, disappointment in love, rankling grievances, and perhaps ungratified sexual desires or masturbation, may contribute to the “neuropathic” origin of chlorosis. A change of climate seems to operate as a cause, and is manifested especially in the case of girls emigrating from Ireland to enter domestic service here (Townsend).

**Symptoms.**—A brief outline of the more frequent and prominent general manifestations of chlorosis—or “green sickness”—may be narrated at the outset. The gradual onset is usually marked by languor, indisposition to either physical or mental exertion, motor weakness, irritability or inertia of mind, depression of energy, and a more or less constant fatigue. *Palpitation of the heart* and *dyspnea* on slight exertion are much complained of in most cases; *headache* is also an early symptom, and may be accompanied by vertigo in some cases; and *dyspepsia* and *constipation* occur in 65 per cent. of cases (Townsend). Probably in one-half of all cases cessation of, or scanty and irregular, menses may form the burden of complaint.

There are also certain special symptoms, among which the following may be mentioned: The appetite is either poor or perverted, and a capricious desire for such innutritious substances as chalk, slate-pencils, and even bits of earth (*pica*), or for sour, highly spiced, and unwholesome articles of food (*malacia*), is not uncommon. Morning vomiting or regurgitation of food and eructations occur in some cases, and dilatation of the stomach and high position of the diaphragm are found in many instances. The tongue is pale, flabby, often dry, and the edges show indentations.

*Constipation* is usually present, though sometimes diarrhea, lasting for a day or two, may alternate, as after the ingestion of some unwholesome article that has been eaten to satisfy the perverted appetite.

The general appearance is distinctive. The subcutaneous fat is not only well retained, but in many cases is even increased, and the rotundity of the body and members preserved. The peculiar *greenish-yellow tint* of the complexion is, however, the most striking manifestation to the eye. It differs thus from the muddy pallor of cancerous anemia, from the lemon-yellow tint of pernicious anemia, from the saffron hue of jaundice, and from the blanched pallor after severe hemorrhages. The scleræ are often pearly- or bluish-white (“cerulean hue”), and, though this is considered by many the earliest positive indication of anemia, when the

skin-tint is not characteristic, yet, according to Townsend's analysis of 87 cases of chlorosis, it is not the most constant. The nails showed pallor in 95 per cent. of the cases; the cheeks, tongue, and lips were pale in 89, 84, and 76 per cent. respectively, while the scleræ were pale in but 64 per cent. On exertion the cheeks and lips may become quite ruddy in cases of moderate anemia (*chlorosis rubra*).

Besides the breathlessness, palpitation, and the tendency to vertigo and syncope complained of in the majority of cases, other circulatory disturbances may occur. The *skin* is often cool, and the extremities are frequently cold, owing to sluggish heart-action. The *pulse* is usually full and easily compressible, and, owing to its excitability, it may be accelerated for the time being by various external influences (see Fig.

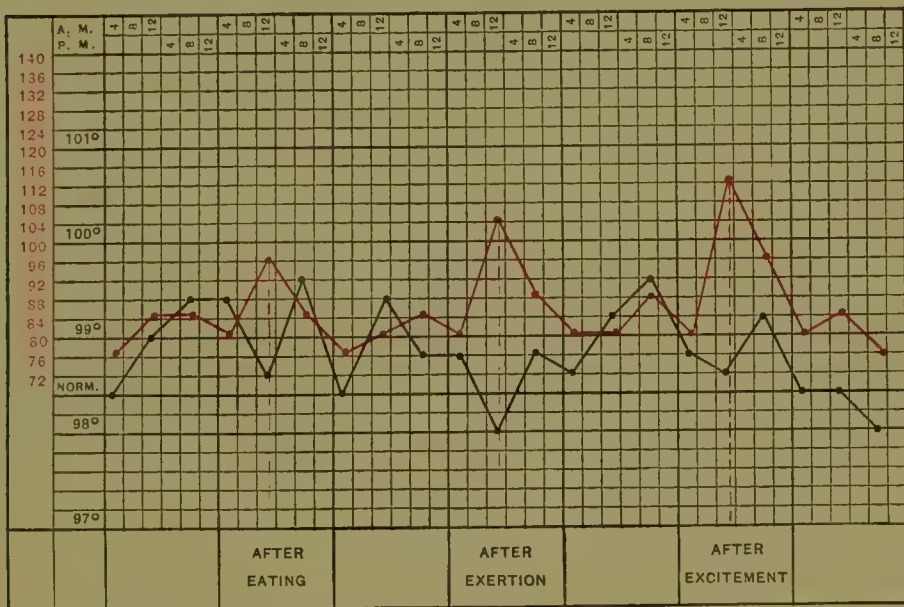


FIG. 33.—Pulse- and temperature-chart of a case of chlorosis, showing the effect exerted upon the pulse by eating, exertion, and excitement.

33). Visible undulating pulsations of the carotid vessels are frequent, and a pulsation in the peripheral veins is also observed at times. Physical examination shows the *heart* to be slightly dilated. Systolic murmurs, soft and “whiffing” in character, are heard at the base, though in severe cases they may be heard at the apex of the heart also. *Systolic blowing murmurs* of hemic origin are not infrequently heard over the carotid arteries. More common and characteristic, however, is the *venous hum* or *bruit de diable*—the soft continuous murmur heard over the large cervical veins. Thrombosis of the larger veins, or of the femoral, or of a cranial sinus, may occur and is always of serious import.

Of the *nervous manifestations* that are often present, neuralgias of the head, mental depression, hyperesthesia of the skin, particularly of the abdomen, gastralgic attacks, and hysteria, are most frequently met with. Tinnitus aurium and anemic amaurosis have been known to occur.

*Edema* of the ankles is found in perhaps one-third of the cases. The *urine* is generally pale, free in quantity, and its specific gravity is somewhat lowered; and according to recent studies there is a diminished



excretion of nitrogen in the form of urea, despite the abnormal destruction of albuminoids.

**Blood-examination.**—The blood flowing from a punctured finger-pulp or ear-tubule is pale, though seldom thin or hydremic, and the paleness is due to a qualitative rather than a quantitative change. There is a disproportionate reduction of the hemoglobin as compared with the number of the red cells. The hemoglobin may range from 50 per cent. to as low as 16 or 17 per cent. in severe cases, the average quantity being about 38 or 40 per cent. On the other hand, the number of red corpuscles is not greatly reduced, and may even be normal. The moderate oligocythemia and marked oligochromemia are almost distinctive of chlorosis: these features, however, may be closely simulated by the chloroanemia of syphilis or early tuberculosis. The average number of red corpuscles is from 3,700,000 to 4,000,000 per cubic millimeter of blood, but the count in very severe cases may be as low as 1,900,000. Approximately, the number of red corpuscles is from 70 to 85 per cent. of the normal, while the leukocytes are only slightly increased in number (8000 to 8500 per c.mm.). Microscopically, the red cells are seen to be paler than normal, and somewhat altered in size and shape. Some are distinctively larger than is usual (macrocytes), but the majority are slightly undersized (microcytes). Irregularity in shape (poikilocytosis) is seen in quite a number of the red cells in the severe cases, and an occasional normoblast (small nucleated red corpuscle) may be noted.

**Diagnosis.**—When the greenish pallor of the face is marked this can often be correctly made at a glance. The blood-examination must be made, however, to completely establish the diagnosis, even when distinctive symptoms are present, such as the shortness of breath, palpitation, weakness and languor, faintness, amenorrhea, capricious appetite, together with a well-nourished appearance of the body. The bluish-white scleræ and pallid nails are confirmatory when observed, and the physical signs should also be sought for.

The primary character of the anemia may be determined in doubtful cases, or in those in which incipient tuberculosis or Bright's disease may be suspected, by exclusion. Here the physical examination of the chest and urinalysis should supplement the blood-examination. Organic disease of the heart may be simulated by the breathlessness, palpitation, vertigo, and edema. According to F. P. Henry, the following blood-variations may be considered in the diagnosis of chlorosis: (1) the red corpuscles may be normal in number and in size, the only change being a deficiency of the hemoglobin; (2) the corpuscles may be normal in number, but diminished in size, while the percentage of hemoglobin is normal; (3) the corpuscles may be diminished in number, with either a diminished, normal, or perhaps an increased percentage of hemoglobin.

**Prognosis.**—This is always favorable, except in those cases in which congenital or developmental anomalies of the vascular system are associated. The discontinuance of proper treatment before a substantial cure is effected is often followed by a relapse, and even after apparent cure one or more recurrences may be witnessed before the age of thirty. The average duration of a case of chlorosis is from two to three months. In cases of very severe type, in which the dividing-line

between this disease and pernicious anemia may not be marked clearly, the prognosis should be made with due reserve.

**Treatment.**—While the treatment of chlorosis by the administration of iron is wellnigh specific, the *hygienic measures* are also important, and particularly in order that relapses may be avoided.

Pure air, wholesome food, and plenty of rest and sleep, with regular habits, are prime requisites. Sometimes a change of occupation, even temporary, where confinement may be replaced by an out-door life, fresh air, and sunshine, as in the case of store-girls and mill-operatives, is of great value in bringing about a rapid improvement. Patients in better circumstances may be sent to rural districts, the mountains, or sea-shore. In cases marked by much palpitation, dizziness, and dyspnea, rest in bed for a week or so is often imperative at the outset. As improvement goes on, however, light and then moderate exercise may be permitted out of doors, and the increasing appetite should be gratified by a generous, easily assimilable diet (milk, meat, eggs, fish, purées of green vegetables, stewed fruit, apples, etc.). Fats and carbohydrates, however, should generally be avoided. Ferruginous mineral waters when procurable may be freely drunk, but coffee, tea, and alcoholics

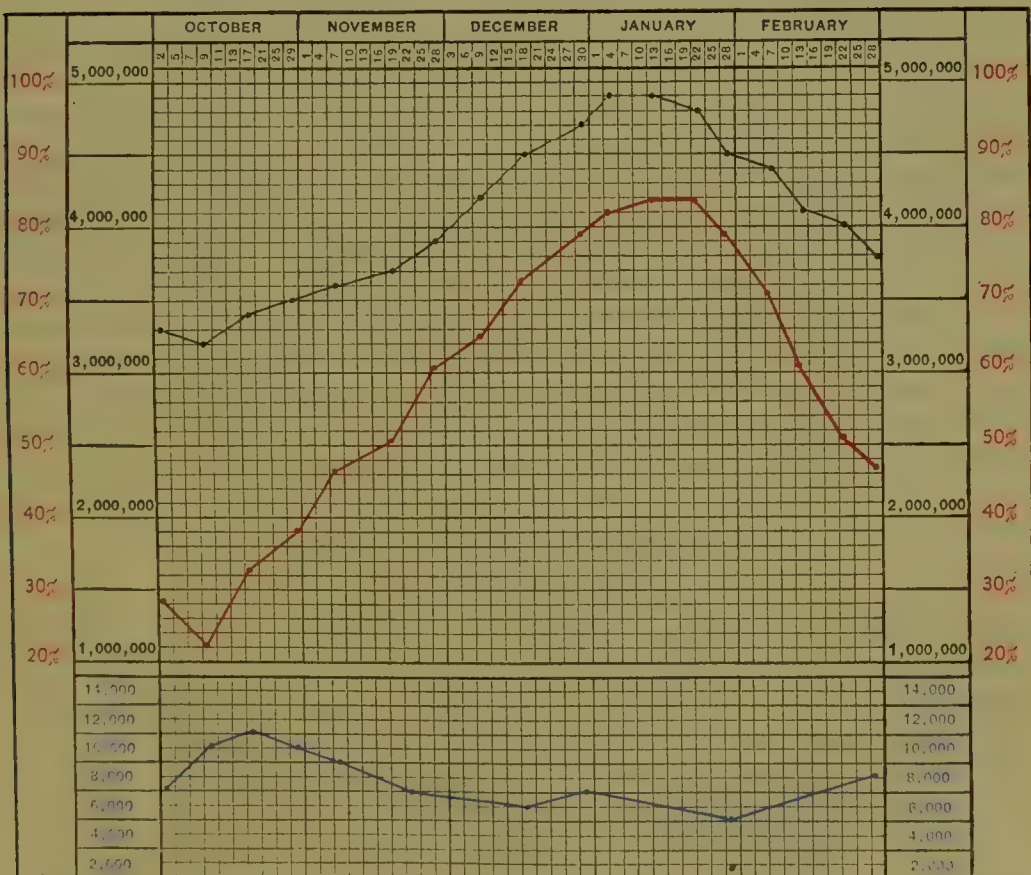


FIG. 34.—Chart of a case of chlorosis, showing the improvement following the administration of iron. Convalescence almost complete; relapse. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

do more harm than good. The one remedy, *par excellence*, on both rational and empirical grounds, is a good preparation of iron. This should be given methodically and persistently, until the percentage of



hemoglobin is 90, and then maintained there by continuing the administration of the iron for several weeks to prevent a recurrence (Fig. 34). Exactly how the iron acts in curing chlorosis has not been definitely proved, but its almost specific action is indubitable. Not all preparations of iron are equally well borne by the stomach, however, and several changes may be necessary during the course of a given case. Probably the best form for general use is the dried sulphate, usually given together with potassium carbonate in the well-known Bland's pills—2 grains (0.129) of each to the pill. Starting with one pill thrice daily for a week or ten days, the daily dosage is increased until nine pills daily are administered in the third week, and continued for several weeks or as long as the case may require. It is very important, meanwhile, that the bowels should be kept soluble by the use of cascara sagrada, salines, and the like. A preliminary course of intestinal antiseptics for a week or so is strongly advised by some authorities, and is worthy of recommendation. Beta-naphthol, thymol, guaiacol, and salol are used for this purpose. The hematinic effect of the iron seems to be produced earlier and better when this plan is followed; and this fact seems to give corroborative evidence to Bunge's theory of the absorption of the iron in chlorosis—in a certain class of cases at least. Other iron preparations of value in this disease are the citrate, protoxalate, lactate, carbonate, the succinate, and the reduced iron. The albuminates of iron, so much vaunted for a time, are practically worthless. In severe cases Quincke uses at first a 5 per cent. solution of the ferric citrate, hypodermically (Mvijss—3ijss—0.5–10.0, daily). The preparation known as ferratin is also highly recommended by some, and the therapeutic efficacy of glycerin extract of bone-marrow in chlorosis is as yet doubtful. Bitter tonics and dilute hydrochloric acid are indicated in a certain number of cases in which indigestion is troublesome. The acid tincture of iron chlorid is sometimes used in such cases. Mild cases often yield to the simple use of remedies for the cure of gastro-intestinal derangement. Adjuvants in the treatment of chlorosis that may be of use are arsenic, manganese, mercuric chlorid, and arsenite of copper in minute doses.

#### PROGRESSIVE PERNICIOUS ANEMIA.

(*Idiopathic Anemia.*)

**Definition.**—A grave blood-disease characterized by a great destruction of red corpuscles, and a persistent tendency from a bad to a worse condition. It usually ends in death, and seldom exhibits causal lesions other than those of the blood or blood-making organs.

The term "idiopathic anemia" applied to this disease by Addison, whose first clear description of its clinical history has become classical, is applicable to a proportionately smaller number of cases to-day than during his time. This is owing to the later discovery (*post-mortem*) of adequate causes for the pernicious anemia that during life could not be found. Thus, while still a primary essential anemia in most cases, and whilst future investigations may show the true Addisonian type of pernicious anemia to be a severe secondary anemia, for descriptive purposes it will nevertheless be convenient to classify both groups under the title of *progressive pernicious anemia* in order to describe the



invariable tendency of both. Under Diagnosis (*vide infra*), however, will be found some differential clinical features.

**Pathology.**—As in chlorosis, the subcutaneous fat is rarely diminished, so that emaciation is exceptional. The skin is pale and of a lemon-yellow tint, and most of the tissues and organs are anemic, except the muscles, which are often decidedly red in color. The fat is usually pale and yellowish, and fatty degeneration is one of the most striking changes in this affection. The heart is usually large and flabby, and on section of the ventricular walls there is a marked pallor, as well as a friability, and a fatty change shown by the yellow tint. Microscopically, the fibers or columns of heart-muscle are seen to be distinctly fatty. The heart-cavities contain very little light-colored blood. Other organs showing the fatty degeneration (of the epithelium) are the liver, kidneys, gastric and intestinal walls, and the intima of many of the smaller blood-vessels (in patches). This general fatty change is probably directly due to the deficient oxygenation of the tissues and to the anemic blood-supply.

Owing to the above degenerative change, and consequent weakening in the vessel-walls, small extravasations of blood are found in different parts. Most frequently these punctiform hemorrhages are seen in the retina and on serous membranes, as on the inner surface of the dura mater, the pericardium, and the pleura. Ecchymoses are also observed occasionally on the mucous membranes and on the skin. More or less general edema and dropsical accumulations in the serous cavities are not uncommon. The spleen and liver are seldom and only very slightly enlarged. The lymph-glands are often somewhat swollen and intensely red in color, owing to the unusual number of red corpuscles, some of which are nucleated.

A marked and important pathologic feature of pernicious anemia is the presence of abundant deposits of iron-pigment, especially in the liver, but also in the spleen, kidneys, pancreas, and other organs. The fact that the abnormal quantity of iron in the liver is peculiarly distributed about the periphery and middle zone of the lobules is particularly noteworthy, and quite characteristic of pernicious anemia. The origin of this iron is doubtless the enormous destruction of red corpuscles, and that the pigment in the hepatic lobules is ferruginous may be determined by a micro-chemic test with ammonium sulphid, granules of black sulphid of iron being formed.

Of special interest are the lesions found in the bone-marrow on account of its hematopoietic function. This is virtually hypertrophied, and is in many cases deep-red instead of yellow, and more like the hemoblastic marrow of childhood (H. C. Wood). Indeed, the fat-marrow of the long bones is often entirely replaced by the red marrow, which makes evident the contrast between it and the icteric pallor of the fatty tissues elsewhere in the body. Cellular hyperplasia may be seen microscopically in the great number of large and small granular medullary cells, and also in the nucleated red cells.

An atrophied and polypoid condition of the gastric mucosa, more or less extensively involving the gastric tubules, is noticed in some cases. The sympathetic ganglion cells may also show changes. More constant, however, is the sclerosis of the posterior columns and, to some extent,

of the lateral columns of the spinal cord: this is especially marked, according to Burr, in the cervical swelling. These sclerotic changes are probably secondary either to the blood-state or to minute hemorrhages.

**Etiology.**—There are three etiologic categories into which cases of pernicious anemia may be grouped: (1) those cases in which no discoverable cause for the hemolysis (blood-destruction) is ascertained, either during life or after death—*i. e.* the idiopathic variety of Addison; (2) those in which an adequate cause is found *post-mortem* only; (3) those that are plainly traceable, *ante-mortem*, to some sufficient primary causal condition acting directly or indirectly.

(1) As regards the *obscure cases* of idiopathic anemia—or cachexia—the essential cause of the symptomatic condition is evidently an actively increased *hemolysis*. The blood-destruction is so great that blood-generation (*hemogenesis*) is overbalanced. The latter may be normal in power or there may be a congenital or acquired underlying deficiency in hemogenetic power; but in either event the hemolysis far exceeds the hemogenesis in pernicious anemia, the liver being the principal seat of the hemolytic changes—in the final stages, at least. Stengel believes that the hemolysis originates in the gastro-intestinal capillaries, and depends upon poisons generated or absorbed from that tract—an auto-intoxication.

(2) *Apparently causeless* cases of progressive pernicious anemia may be found *post-mortem* to have been caused by (a) obscure malignant disease; (b) parasites, especially the *Anchylostoma duodenalis*, and rarely by the *Bothriocephalus*. Not infrequently, by a careful study of the anamnesis of a patient, aided by modern methods of examination, the cause of pernicious anemia may be detected during life. Atrophy of the stomach and chronic gastritis, with polypoid growths of the mucosa, may be included in this category. The *Bothriocephalus latus* may be discovered during life, though more frequently only after death.

(3) Certain exhausting causes, operating directly or indirectly, may precede this affection, as severe or prolonged hemorrhages, or diarrhea, fevers, mental shock, profound chlorosis, pregnancy, and parturition.

Unfavorable hygienic surroundings and insufficient nourishment, habitually kept up, may also favor the development of the disease; but, as in chlorosis, the most favorable environment is not by any means preventive of its development. Males are more frequently affected than females, and especially does it occur during middle life, though occasionally cases are seen in those below twenty years of age. The disease is widely distributed, and, whilst it has been observed to behave almost endemically at times, as in Switzerland and Leipsic, no infectious origin has been shown to exist.

**Symptoms.**—Idiopathic pernicious anemia develops so slowly and insidiously that it is hardly ever possible to fix upon any precise date as the commencement of the disease. The transition from health to progressive pernicious anemia, particularly in persons previously feeble and pale, is usually too gradual to be demonstrable; though a rapid and acute onset is rare, it may occur in pregnant or puerperal women.

*Pallor* is soon noticed and gradually increases, or when there has been a previous pallor, this becomes more marked. *Shortness of breath* and *palpitation of the heart*, especially on exertion, are complained of;



the patient is also easily fatigued, and becomes quite languid. Occasional nausea may come on early in those cases in which a previous gastro-intestinal disturbance has been noted, and headache, vertigo, tinnitus aurium, and anorexia ensue and grow progressively worse. General weakness increases, and occasional attacks of faintness and vomiting supervene. Meanwhile, the skin takes on a bloodless, waxy appearance, and soon the characteristic *lemon-yellow tint* appears. The mucous membranes (lips, gums, conjunctivæ) are likewise pale and colorless. *Prostration in bed* gradually becomes almost absolute as the feebleness and flabbiness of the tissue increase. *Malleolar edema* is sometimes noticable, and ecchymoses—mucous and cutaneous—though not so common as retinal hemorrhages, are seen in profound cases of anemia. Although the intellect is not impaired, except that mental exertion becomes irksome, the tone and manner of speech are feeble, slow, and apathetic. As the debility becomes severe the mind wanders, and, to use Addison's words, the patient "falls into a prostrate and half-torpid state, and at length expires."

Emaciation is rare, the fat being preserved and sometimes increased in quantity. Pulsation in the large arteries is abnormally visible, and a diffuse, exaggerated cardiac impulse is felt. The *pulse* early in the case may be strong, and generally it is rapid (100–120), soft, and compressible, and as full and quick, often, as the water-hammer pulse of aortic regurgitation. Auscultation reveals the constant and characteristic *hemic murmurs*, best heard at the base of the heart, and the *bruit de diable* in the veins of the neck. There may also be visible pulsations in the latter.

*Gastro-intestinal symptoms* may be the most prominent signs in cases where gastritis polyposa and gastritis atrophica are causal. Diarrhea, dyspepsia, nausea, and vomiting are then present throughout the long course; otherwise, constipation, eructations, and simple anorexia are most common.

An ophthalmoscopic examination shows the cause of the anemic amaurosis, in the profound cases of anemia, to be one or more retinal hemorrhages. The liver and spleen are rarely palpable. The bones, and especially the sternum, are sometimes sensitive to pressure.

**Respiratory Symptoms.**—The breathing is accelerated, and the anemic dyspnea may become very pronounced and stertorous, accompanied by a sense of oppression in the chest and a "hunger for air." Near the end pleural and pericardial serous effusions and pulmonary edema tend to appear.

The *urine* is of low specific gravity, and, on account of its pigmentation with pathologic urobilin, dark in color. The urobilin is detected both by chemic and spectroscopic examination. In the former the addition of a few drops of an alcoholic solution of zinc chlorid to the urine gives a green fluorescence. Peptonuria is of doubtful significance. Albumin and glucose are absent, but uric acid and urea are both increased in amount, the former occasionally and the latter usually.

*Fever* of a moderate degree is commonly, though not invariably, present, the evening temperature sometimes reaching 102° F. (38.8° C.). Previous to death the temperature may be subnormal.

**Nervous Symptoms.**—Paresthesia, spastic paralysis of the limbs, and



a loss of control of the sphincters indicate the paralytic tendency of those cases in which sclerosis of the cord occurs. Tabetic symptoms are sometimes marked.

**Blood-examination.**—The blood is usually pale, though sometimes dark and watery, and the oligocythemia is distinctive of pernicious anemia. The number of red corpuscles may be reduced to less than 200,000 per c.mm., and is seldom more than 1,000,000; in severe cases about half a million is the usual count. There is ordinarily no increase in the number of leukocytes; on the contrary, they may be somewhat diminished. The percentage of hemoglobin may be approximately proportionate to the number of red corpuscles, but more often it is relatively increased, so that the individual corpuscles are rich in hemoglobin. In other words, although there is a reduction in the total amount of hemoglobin, it is usually not so great as the reduction in the number of erythrocytes; therefore, the percentage of hemoglobin is nearly always relatively higher than that of the red globules (see Fig. 35), a condition

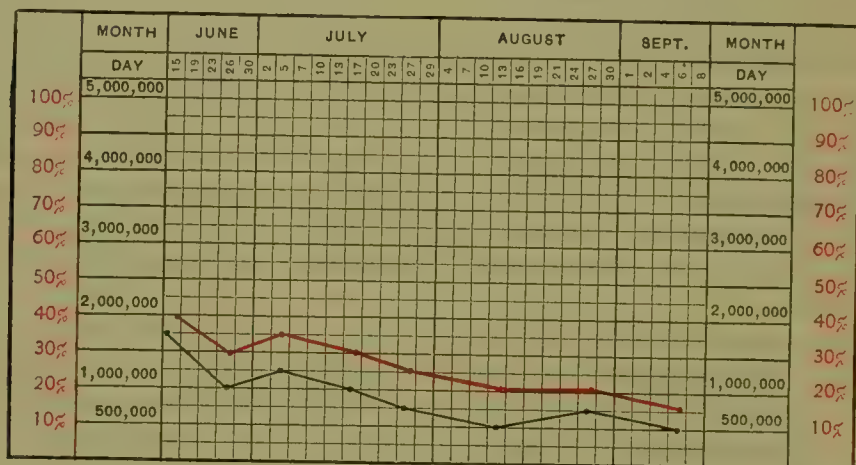


FIG. 35.—Blood-chart of a case of progressive pernicious anemia. Black, red corpuscles; red, hemoglobin.

in marked contrast with chlorosis. Macrocytes, microcytes, and poikilocytes are abundant, and the macrocytes are supposed to give rise to the relatively larger percentage of hemoglobin. The presence of nucleated red corpuscles is also a striking characteristic of pernicious anemia. When normal in size they are known as *normoblasts*; when very large, as *gigantoblasts*. In the former, according to Ehrlich, the eccentrically-placed nuclei stain deeply; in the latter the large nuclei stain faintly. The former are typical of those nucleated red globules found in the hematopoietic organ of adults; the latter, of those found in the blood-development of embryonic life. The gigantoblasts are numerous in this disease. There are other and various forms of degeneration of the red cells, but these are of minor import. There may be an increase in the small lymphocytes at the expense of the polynuclear cells; and, according to Cabot, the presence of large numbers of polychromophilic red cells has been noted in a series of 50 cases. The blood-plates are generally fewer than normal. The relative proportion of the proteids in the blood-plasma is altered (Adami).

**Diagnosis.**—It is important to determine, if possible, whether the

anemia is truly primary (or idiopathic) or secondary. Moreover, the possibility of hidden carcinoma, gastric atrophy, the anchylostoma or other parasite, and incipient tuberculosis should be borne in mind. *Intestinal parasites* may be inferred from the microscopical examination of the feces after a brisk purge if the eggs of the parasites or the parasites themselves be found. *Atrophic gastritis* may be discriminated by examining the viscus and gastric juice by modern methods. The following table will permit the elimination of obscure gastric carcinoma:

PROGRESSIVE PERNICIOUS ANEMIA.	OBSCURE GASTRIC CARCINOMA.
The blood shows characteristic changes, and the red corpuscle count falls to or below 1,000,000 per c.mm.	Blood shows characteristics of secondary anemia, and the count does not fall to 1,000,000, as a rule.
Found earlier in life.	Occurs after middle life.
Gastric symptoms not so prominent.	Gastric symptoms more suggestive.
Lemon-tinted skin common.	Skin of a pale, muddy-color, or only slightly jaundiced (saffron-yellow).
Adipose tissue fairly well preserved.	Progressive emaciation.
No glandular enlargements palpable.	Supraclavicular or inguinal glands may be palpable.
No physical signs over stomach.	There may be an area of increased resistance over the stomach.
Examination of gastric contents after test-meal usually negative.	Examination of gastric contents shows deficiency or absence of free hydrochloric acid and presence of lactic acid.
Some improvement may be brought about—even cure, though very rarely.	Condition becomes steadily worse until death ends the case.

From *chlorosis* the affection may be differentiated easily by the blood-examination. The relative increase in hemoglobin, the presence of giantoblasts and many macrocytes, and the severe oligocythemia are pathognomonic of pernicious anemia, and are in marked contrast to the oligochromemia, and slight, if any, reduction in the number of red globules of chlorosis. Again, the progressive pernicious character of the former and the tendency to hemorrhage should be remembered, as well as the contrasting factors of age and sex in the two affections.

**Prognosis.**—The disease, as a rule, terminates fatally, though not so frequently now as at one time, for obvious reasons. The course of pernicious anemia is usually slow and gradual, and may be interrupted by improvement or apparent recovery. Recurrences, however, are prone to occur, even after intervals of several years, “attacks of anemia” alternating with periods of improvement, accompanied by enlargement of the spleen. Idiopathic anemia is therefore almost hopeless, although a few apparently substantial recoveries have been reported. The duration of the disease is seldom more than a year, and may not be more than two or three months. Death may be caused either by syncope, cerebral hemorrhage (most commonly), or by slow asthenia.

**Treatment.**—**Hygienic measures** must be regarded as of signal importance, and rest in bed, together with light nutritious food given at short regular intervals, is indicated first of all. Salt-water baths and gentle and systemic massage when the patient is at absolute rest and is not too weak, are useful adjuvants.

The value of *arsenic* in progressive pernicious anemia is analogous to that of iron in chlorosis. The best action of the drug will be ob-

tained by the administration of gradually ascending doses of Fowler's solution or of arsenous acid. Beginning with four or five drops of the former, three times daily during the first week, and thereafter adding one drop to the dose every day or two up to the point of tolerance, as much as twenty or thirty drops, well diluted, may be taken (see Fig. 36). Evidences of gastro-intestinal irritation should be watched for,

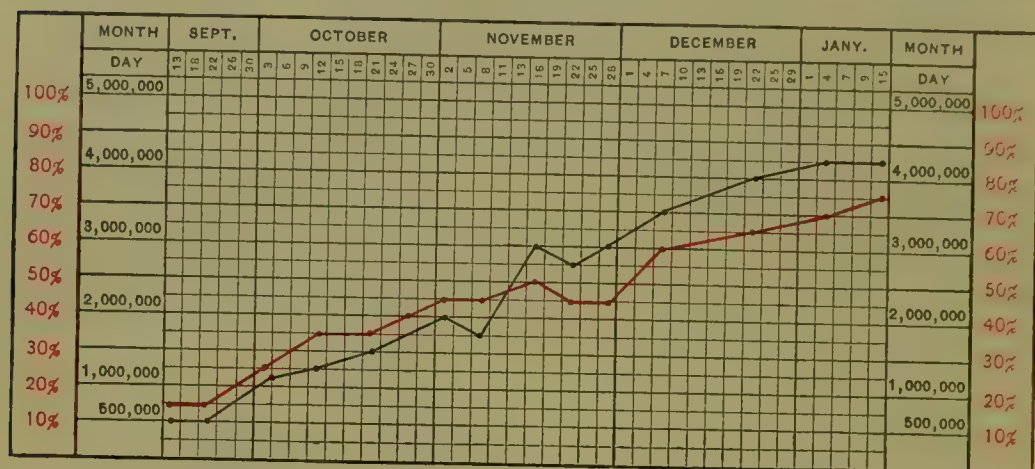


FIG. 36.—Chart of a case of progressive pernicious anemia, showing the improvement following the administration of arsenic. Black, red corpuscles; red, hemoglobin.

and the arsenic either discontinued or the daily dose reduced should they appear. Sometimes it is advisable to use the remedy hypodermically. Arsenous acid is given in pill form, commencing with  $\frac{1}{30}$  or  $\frac{1}{20}$  gr. (0.0021–0.0032).

The introduction by Fraser of Edinburgh of bone-marrow in the treatment of pernicious anemia has been followed by various results: some cases have been reported in Great Britain and in the United States in which it has seemed to do good, while in others it was found to be useless. While the glycerin extract is the preparation generally used, it is not so reliable as the raw red bone-marrow, or that freshly prepared each day by mixing with it an equal quantity of glycerin; an ounce or two may be administered daily. The remedy is worthy of trial, and if found to be non-efficacious in the given case, arsenic may either be combined with it or used alone.

Near the end of the disease the danger often greatly increases, owing to the marked reduction in the quantity of the blood (*oligemia*). This may be combated by the injection of warm water or a weak saline solution into the colon (enteroclysis) and also into the subcutaneous tissue (hypodermoclysis). Both the former procedure and gastric lavage are of value in preventing and ameliorating the gastro-intestinal disturbance from fermentation and putrefaction. *Intestinal antiseptics* (thymol, guaiacol carbonate, salol, beta-naphtol, and hydro-naphtol) should be given by the mouth in conjunction with the injections, and lavage of the tract should be employed for the same purpose.

Anthelmintics must be used in those cases of pernicious anemia in which intestinal parasites are associated. Dilute hydrochloric acid, nuxvomica, and bitter tonics are serviceable in cases in which gastric digestion is impaired.



During the convalescence in favorable cases iron seems to be peculiarly valuable, sometimes alone and frequently in conjunction with arsenic. Thus, arsenious acid and either the carbonate of iron or reduced iron may be combined in pill form, or Fowler's solution and the tincture of the chlorid of iron, or Blaud's pill, may be used with satisfactory results. Recurrences will yield to the same treatment, if they yield at all, except that the doses may have to be increased according to the tolerance of the individual case.

## II. THE SECONDARY ANEMIAS.

The secondary anemias are symptomatic of abnormal processes or of existing disease, whether acute or chronic, and their causes are numerous and various. I have already stated that secondary anemia may occur when the true primary form cannot readily be determined and when the course of the anemia is progressive and pernicious. Furthermore, several possible causes may exist in a given case of symptomatic anemia, and it may be quite difficult to discover which of these is the active factor in the condition.

In certain secondary anemias, also, the associated impairment of the blood-making organs is so evident that the anemia may assume almost a primary importance. This was exemplified in Strümpell's case of carcinoma and anemia, with secondary implication of the bone-marrow. The variety and uncertainty of the causes of secondary anemias thus prevent a satisfactory classification.

**The Blood.**—In most cases this distinctly differs in character from the blood of the primary or essential anemias. There is oligocythemia, usually of a moderate degree, about 3,000,000 red corpuscles per cubic millimeter being noted, though in cases of severe hemorrhage the reduction may be as great for a time as in pernicious anemia. There is also a relative decrease in the amount of hemoglobin, and sometimes the percentage may be relatively lower even than is compatible with the decrease in the number of the red corpuscles. There is a relative, and often an absolute, increase in the number of leukocytes (*vide* Fig. 37). Either a few or many poikilocytes, a few macrocytes, microcytes, and normoblasts are found, depending upon the severity of the anemia. Gigantoblasts are not seen, and the relative increase in the percentage of hemoglobin is also absent in secondary anemia.

The most important etiologic groups of secondary anemias are as follows: (1) **Hemorrhage.**—Hemorrhages occur under a great variety of circumstances, and if copious result in an acute secondary anemia. Thus there may be the rupture of an aneurysm, menorrhagia, postpartum hemorrhage, hemoptysis, gastrorrhagia, enterorrhagia, etc., all of which produce the same general effect upon the system. Repeated small hemorrhages may finally produce the same result as a single large one, and spontaneous hemorrhages or epistaxes, such as occur in persons of a hemorrhagic diathesis (hemophilia) or in purpura and scurvy, may cause profound secondary anemia. Females are most tolerant of losses of blood, but infants of both sexes bear depletion very badly. The total mass of blood may be much diminished (oligemia), and the sudden loss of a great volume of blood may prove fatal in a few moments; but

it is often surprising how recovery can take place, and often does, after the rapid loss of several pounds of blood—*e. g.* in hemoptysis, hematemesis, or menorrhagia. Sometimes the source of bleeding is obscure, as in cases of intestinal parasites, hepatic cirrhosis, or duodenal ulcer; or it may be intentionally kept *sub rosa* by females having uterine disorder or bleeding hemorrhoids. The quick blanching of the countenance, the weakness, the coldness of the skin, faintness, dimness of vision, tinnitus aurium, sighing respiration, and feeble, rapid pulse are characteristic symptoms of acute anemia. *Unconsciousness and epileptiform*

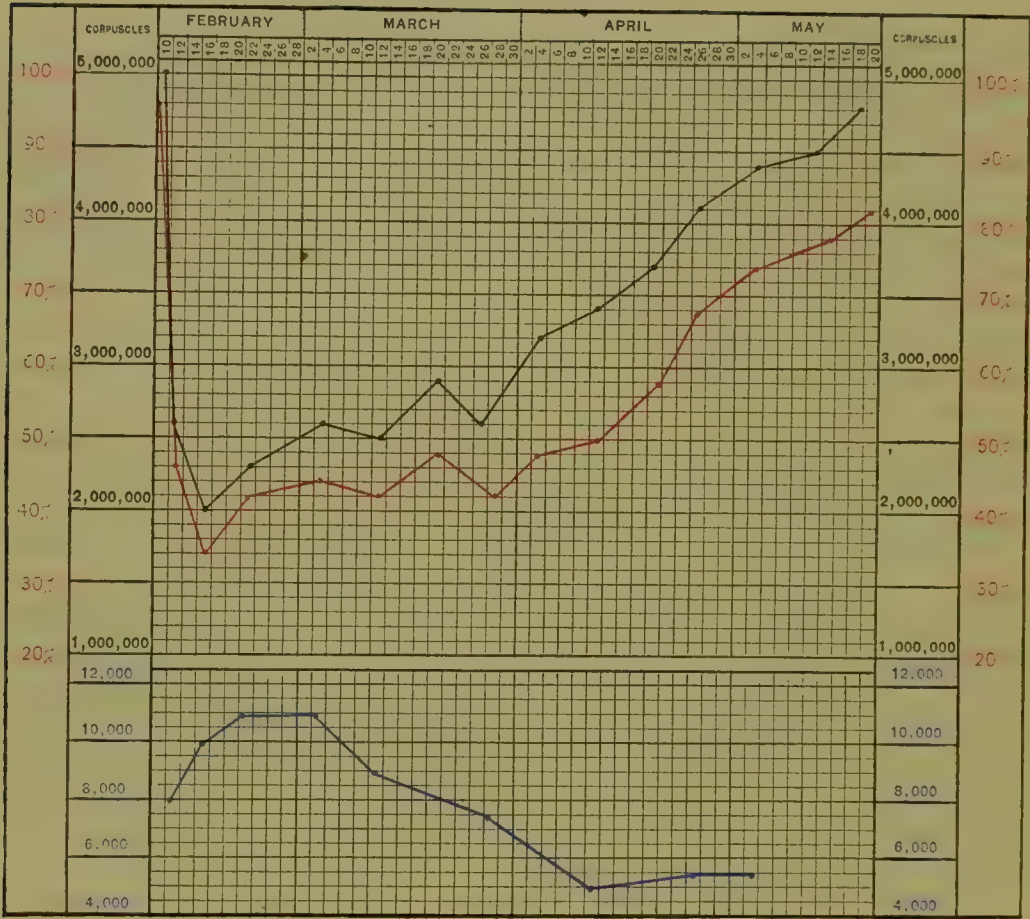


FIG. 37.—Blood-chart of a case of symptomatic anemia. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

*convulsions* precede death in cases in which the total volume of blood lost is sufficiently large. When recovery takes place the blood-regeneration goes on rapidly, so that within from one to three weeks restitution is complete. The normal volume is soon restored—first by the absorption of water, hydremia existing for several days before the saline and albuminous elements are renewed. The white corpuscles are earlier restored than the red, so that there is a temporary relative leukocytosis. The hemoglobin is restored still more slowly than the red corpuscles.

(2) **Inanition.**—Anemia from inanition may be caused by a food-supply that is insufficient either in quantity or quality, or both; or, even with abundant food of sufficient nutritive qualities the digestive power may be so impaired as to cause defective assimilation. Esophageal

carcinoma and chronic gastritis, especially of the atrophic variety, may thus cause anemia from inanition. The reduction of the blood-plasma forms a feature, while the corpuscles may be affected but slightly.

(3) **Excessive albuminous discharges**, as in chronic Bright's disease, prolonged suppuration, long-continued lactation, chronic dysentery, etc., drain the system so that marked anemia may be produced.

(4) **Toxic Agents**.—The poisons may either be organic or inorganic, though toxic anemias are most common from the absorption of lead, arsenic, mercury, and phosphorus. The poisoning is usually chronic, and affects principally the corpuscles. Anemia due to the poisons of acute or chronic infectious diseases is also frequently met with, and may thus be observed after typhoid fever, diphtheria, yellow fever, and inflammatory (articular) rheumatism among the acute diseases, and during chronic malaria, tuberculosis, and syphilis ("syphilitic chlorosis"). There is considerable destruction of the red corpuscles in some of these diseases, either directly or indirectly, and the greater the pyrexia the greater the action upon the blood or blood-making organs.

**Symptoms**.—The common indications of secondary anemia are the pallor of the face and mucosæ, muscular and mental weakness, loss of nerve-function, neuralgias, coolness of the skin, dyspnea on exertion, cardiac palpitation, impaired appetite and digestion, and a weak pulse. The physical signs are those of the primary or essential anemias.

**Diagnosis**.—Here may be advantageously contrasted the distinguishing features naturally grouping themselves under symptomatic and essential anemias, respectively :

SYMPTOMATIC OR SECONDARY ANEMIA.	IDIOPATHIC OR ESSENTIAL ANEMIA.
A symptomatic blood-condition secondary to disease elsewhere. Occurs at any age.	A primary disease of the blood and blood-making organs. Occurs principally during adolescence and early middle life.
Previous or associated history of traumatic or spontaneous hemorrhage, chronic suppuration, prolonged lactation, chronic Bright's disease, carcinoma, chronic lead-poisoning, chronic malaria, etc.	Previous history negative in its bearings upon the disease.
Blood-changes not so marked and more variable.	Distinctive blood-characteristics, and often profound changes, both as to blood-cells and hemoglobin.
Moderate reduction in both, merely the relative proportion being maintained.	Marked reduction in either the hemoglobin percentage or in the number of red corpuscles.
General symptoms and signs usually subordinate in manifestation to those of the primary disease or lesion.	General symptoms and signs also more characteristic of the respective form of anemia in the case.
Gravity of anemia depends on that of the primary disease.	Gravity depends on type of blood-changes and progressiveness of disease.
Often responds to treatment, depending on the cause; in a few instances, as in hemorrhage, it is short in duration.	One variety (chlorotic) quite curable, the other (progressive pernicious) commonly fatal.

The **prognosis** depends upon the cause of the anemia.

**Treatment**.—Symptomatic anemia is amenable to treatment accord-



ing to the cause. The traumatic acute variety does well under simple hygienic measures after the urgent indications have been met. Plenty of pure air, wholesome food, and graduated rest and exercise may suffice, and drugs not be needed. Cases in which it is difficult or wellnigh impossible to remove the cause of the anemia of course do not improve under any treatment other than that which may favorably influence the primary affection. Nutritious aliment, iron in some form, a judicious hygienic regimen calculated to increase the assimilation, and stomachic and general tonics are required in the majority of cases. Toxic substances must be eliminated, their re-introduction into the body prevented, and the repair of the blood and tissue actively promoted.

#### LEUKOCYTOSIS.

**Definition.**—A temporary increase in the number of leukocytes in the blood, especially of those of the polynuclear variety. These in normal blood constitute about three-fourths of all the leukocytes. The number of white corpuscles in a moderate leukocytosis would be about 10,000 per cubic millimeter; in marked leukocytosis there might be as many as from 20,000 to 40,000; a count of over 50,000 leukocytes to the c.mm. may, however, usually be considered to indicate leukemia. Von Limbeck, notwithstanding, reported a case of leukocytosis accompanying carcinoma of the kidney with metastasis, in which there were 80,000 white corpuscles per cubic millimeter.

*Physiological leukocytosis* occurs in infants during the first few days after birth, in pregnancy, during digestion, and after exercise. According to Carter, the "digestion leukocytosis is present after a meal of proteids or hydrocarbons, but not after a meal of carbohydrates."<sup>1</sup> Massage and cold baths also produce leukocytosis, probably by stimulating the circulation, and not by increasing the actual number of leukocytes, some of which have simply become stagnated.

*Pathological leukocytosis* is secondary to various affections. It may be temporary, as in the curable primary diseases, or permanent, as in those that do not permit of recovery. It is also found to be well marked in acute inflammations and in infectious febrile diseases accompanied with exudation, such as pneumonia and diphtheria. In pleuritis, peritonitis, pericarditis, erysipelas, and in all suppurative processes there is an excess in the number of polynuclear neutrophiles. Inflammations of the serous membranes, when not tuberculous, cause leukocytosis, so that a purulent meningitis may be differentiated from tuberculous meningitis by the pronounced leukocytosis in the former and its absence in the latter. As a rule, the greater the local reaction and the stronger the resistance to severe infections the greater the leukocytosis. As is well known, the pus-cells of an abscess consist almost wholly of dead white corpuscles—phagocytes—that have been overcome or exhausted, directly or indirectly, in the struggle against the toxin of the infection. Cachectic states, as in cases of malignant tumors, are often attended with an increase in the number of colorless corpuscles in the blood, especially in the region of the tumor and where the lymph-glands are involved. Leukocytosis may be very marked in carcinoma,

<sup>1</sup> *Univ. Med. Magazine*, vols. vii and viii, p. 181, Dec., 1894.

the ratio of reds to whites being, in some cases, 25 to 1. Chemical irritants, such as turpentine, may also produce leukocytosis, and whatever the substance causing the condition it is spoken of as *positively chemotactic*—attractive to the white blood-corpuscles—in contradistinction to *negatively chemotactic substances*, which repel the white corpuscles.

In non-leukocytotic infectious diseases, such as typhoid fever, the diagnosis of a complicating pleuritis, for example, may be confirmed, even at its onset, by the detection of the leukocytosis. Leukocytosis under such circumstances has prognostic importance. Diminishing leukocytosis during the height of a grave disease may be significant of lessening powers of resistance, though this is not an invariable rule, since just before the crisis of a pneumonia or when there is marked emaciation, as in typhoid fever, a diminution of the leukocytes is apt to occur.

The object of the leukocytosis is naturally protective, beneficent, and reparative. It is accomplished either by direct antagonism or by the formation of substances that enter the fluids and tissues of the body, and counteract the influence of the toxic substances causing the disease. The existence of leukocytosis can best be determined by the examination of stained specimens of the blood. Physiological digestion leukocytosis is to be discriminated from the pathological variety by making the examination several hours after the last meal has been taken.

#### LEUKOCYTHEMIA.

(*True Leukemia.*)

**Definition.**—A blood-disease, usually chronic, characterized by a peculiarly marked and persistent increase in the number of leukocytes, associated with lesions occurring either respectively or unitedly in the spleen, bone-marrow, and lymphatic glands.

**Pathology.**—Bodily emaciation and pallor are pronounced, and edema, with dropsical effusions in the serous cavities, is by no means uncommon. The cardiac chambers and principal veins are distended with large blood-clots of a greenish-yellow or, in extreme cases, yellowish-white, purulent appearance. Subserous ecchymoses of the pericardium and endocardium are frequent, and the myocardium is often found to have undergone a moderate degree of fatty degeneration. Various abnormal substances have been found in leukemic blood, and among them the following may be mentioned: hypoxanthin, leucin, tyrosin, acetic, formic, and lactic acids, and certain albuminous substances (deutero-albumose and nucleo-albumin) resulting probably from the destruction of blood-corpuscles. The alkalinity and specific gravity of the blood are both diminished. The minute, colorless, octahedral, so-called Charcot's, crystals are found most abundantly in settled leukemic blood, and have also been detected in the spleen, bone-marrow, and liver, as well as in other affections. Their composition is not clearly known.

Although the spleen, bone-marrow, or the lymph-glands may alone show the pronounced pathological changes of leukemia, it is usual to find all more or less affected. Purely splenic or myelogenic leukemia, and the latter especially, are rarer than the lymphatic type, so that it is customary to speak of two principal groups: (1) *splenic-myelogenous* (or *spleno-medullary*) leukemia, the most frequent variety; and (2) lymphatic leukemia.



There is nearly always some splenic enlargement, and in many cases the enlargement is considerable, as in **spleno-medullary leukemia**. Leukemic spleens sometimes weigh as much as from eight to eighteen pounds, and their lengths may vary from six to twelve inches. The enlargement is generally uniform, and the notches upon the anterior border may be much exaggerated. White patches of perisplenitis and a thickened capsule adhering to the surrounding organs and the abdominal wall may also be noticed. The consistence of the spleen is firm and resistant to the knife, though in the earlier stages it may be quite soft and pulpy. The cut surface is either of a uniformly brown color or mottled by the presence of grayish- or yellowish-white circumscribed lymphoid tumors, or by deep-red or brownish-yellow hemorrhagic infarcts. The Malpighian bodies may or may not be visible. The blood-vessels at the hilum are enlarged. Microscopic examination shows the change to consist in a true hyperplasia of the organ, there being an increase in all the normal histological elements. The cells of the pulp sometimes show granular and fatty degeneration, and in advanced cases the trabeculæ may be thickened by a considerable amount of firm connective tissue.

In the majority of cases the bone-marrow is affected as well as the spleen, and a purely myelogenous leukemia is extremely rare. Indeed, the few reported cases of the latter may be doubted. The medullary substance, instead of being fatty, is rich in lymphoid and blood-cells in various stages of development, and is either reddish-brown or greenish-yellow in color. Neuman regarded the marrow-change as a constant and essential lesion of leukemia, and called the former transformation "lymph-adenoid," and the latter "pyoid." The pus-like marrow and the dark-red may exist side by side, although the former is more common.

A fine reticulum may be seen between the cells, especially in the dark-red variety, and small hemorrhagic infarcts may also be noted occasionally. Microscopically, the medulla contains an abundance of lymphoid cells and nucleated red corpuscles. Eosinophile, mononuclear, and polynuclear leukocytes are also present, the first-named being quite numerous, as are also certain myelo-plaques and cells showing karyokinetic figures. The *lymphatic glands* are more or less enlarged in the splenic and medullary forms of leukemia.

In the **lymphatic variety**, especially when acute, an early and marked hyperplasia of all the glands takes place. The cervical, axillary, inguinal, and mesenteric glands are usually involved, and may form distinct, soft, and movable tumors, their color being a reddish-gray, and section often showing hemorrhagic points.

The histological examination shows an increase in the cellular elements. A similar hyperplasia occurs in those glandular tissues that are allied to the lymphatic glands, such as the tonsils, lymph-follicles, the tongue, mouth and pharynx, thymus gland, and the solitary and Peyer's agminated intestinal glands.

The *liver* may be greatly enlarged; indeed, some of the instances of greatest enlargement of this organ have been those due to leukemia, the weight being as much as fourteen pounds. The enlargement is uniform and due to a diffuse leukemic infiltration. The capillaries and interlobular tissue are distended with leukocytes, and disseminated whitish



or grayish nodules, usually quite small, consisting of lymphoid cells undergoing indirect division of their nuclei, are frequently found. Sometimes these leukemic nodules appear as definite growths, with an adenoid reticulum between the cells, on account of which they have been called lymphomata or lymph-adenomata.

Similar changes are observed in the *kidneys*, enlargement, paleness, and diffuse and circumscribed leukemic infiltration of the capillaries and intertubular tissue all being noted. Leukemic nodules may also be found in other parts of the body, such as the retina, brain, serous membranes, lungs, testicles, and skin. Karyokinetic figures are numerous in the cells accompanying these leukemic growths.

**Etiology.**—The primary cause of leukemia is unknown; that it directly affects the blood-forming organs, however, is most probable, though with differences of selection and co-ordination and with different degrees of intensity. The combination of lesions in the spleen, lymph-glands, and bone-marrow, along with the histological similarity of the leukemic growths to the infectious granulomata, and the clinical history of cases of acute leukemia, would seem to point strongly to the *microbic origin* of the disease. Moreover, various cocci and bacilli have been found, but not one of them has been definitely proved to be the specific cause of the disease. *Auto-intoxication* by toxic albuminoids from the digestive tract is believed by Vehsemeyer,<sup>1</sup> who analyzed 600 cases, to be the important point of departure of the disease. It is likely that the direct cause of the leukocythemia is a simple increase of the cytogenic function of one or more of the hematopoietic organs. Kottnitz held leukocythemia to be a reactive condition following auto-intoxication with peptones, and consequently a leukolysis, the over-action of the hematopoietic organs leading to hypertrophy. Whether the reduction of the erythrocytes is due to diminished production or to increased destruction is not positively known, although the former factor is more probably operative.

The disease has often been preceded by an *injury* or a *blow* in the splenic region, but its direct traumatic origin is hypothetical only. *Intestinal ulceration* has been a frequent feature prior to leukemia, and undoubtedly affords a source of possible infection from the tract. *Stomatitis* also may furnish a means of entrance for the infectious agent. The causal relation of *pseudo-leukemia* and *true leukemia* is uncertain, although a few cases of the one have been observed to pass into the other.

In a considerable proportion of cases leukemic patients have had *malaria* of some form. Syphilis may be associated with the disease, but it is not probable that it acts in a causative manner.

*Hereditary influences* undoubtedly play a part; a "lymphogenous diathesis" may thus be transmitted, and several generations may be affected by the disease. *Adverse hygienic* and *social conditions* may also predispose to leukemia. It may also develop after *pregnancy*, or more commonly at the *climacteric*. Anxiety, worry, and mental depression have been mentioned as predisposing causes, with doubtful justification.

Leukemia occurs most frequently in males during the middle period of life, and is apt to attack young persons. It has occurred during infancy, and as late also as the seventieth year, but the average age ranges

<sup>1</sup> *International klin. Rundsch.*, Vienna, Nov. 25, 1894.

from twenty-five to forty-five years. Sometimes the previous condition was one of apparently perfect health.

**Symptoms.**—**Acute leukemia**, although comparatively rare, may be described briefly first. It usually occurs in an adolescent who may have enjoyed previous good health. Its onset is sudden, and usually begins with prostration, hemorrhage of the mucous membranes, and high fever. Acute splenic tumor rapidly develops; the lymphatic glands may enlarge; and palpitation, dyspnea, and gastro-intestinal symptoms of a severe type appear. The blood shows a marked increase in the number of leukocytes, the ratio to the red corpuscles being 1 to 30 or 1 to 50, instead of the normal 1 to 350 or 1 to 600. In acute lymphatic leukemia the lymphocytes are very numerous. Large mononuclear leukocytes and myelocytes are also numerous, while the eosinophilic cells are few in number compared with those found in the blood of chronic leukemia. The case grows progressively worse; hematemesis, cerebral or retinal hemorrhages, and petechiæ supervene perhaps, and the clinical features may then resemble an infectious disease with hemorrhagic and purpuric manifestations.

In **chronic leukemia** the onset is generally slow and insidious and its development imperceptible, and the earlier symptoms may not differ from those of simple anemia for many months. Languor, a deranged appetite, dizziness, noises in the ears, faintness, breathlessness on exertion, and palpitation may all appear. Sometimes, however, not even these symptoms are present, common as they are to most anemic cases, and the patient may first consult the physician, because of a swelling or distress in the left side of the abdomen—the enlarged spleen. Early manifestations may be hemorrhagic in some cases (epistaxis, hematemesis, enterorrhagia), with nausea, vomiting, and diarrhea; or increasing pallor of the countenance or troublesome priapism may be the first indication. As the disease progresses the anemia becomes more marked, edema of the dependent portions of the body may appear, and fever, though slight at first ( $99.5^{\circ}$  F.— $37.5^{\circ}$  C.), may gradually rise to  $102^{\circ}$  or  $103^{\circ}$  F. ( $39.4^{\circ}$  C.), either remaining constant or alternating with periods of apyrexia.

The *pulse-rate* is increased; in quality it is soft and compressible, though sometimes full in volume. The *dyspnea* may be aggravated by the hydrothorax of a general dropsy in advanced cases, or by the upward displacement of the diaphragm owing to the increasing splenic and hepatic enlargement. *Epistaxis* may become obstinate. Bleeding from the gums into the retina (leukemic retinitis) and brain, as well as from the mucous membranes, is common. Hemic murmurs are quite constant.

Ulcerative processes in the bowels may give rise to severe *dysenteric diarrhea*. *Ascites* is usually present in advanced cases on account of the splenic tumor, or owing to pressure upon the portal vein by enlarged glands. *Jaundice* is an occasional event. *Leukemic peritonitis* may occur from the presence of lymphomatous growths in the membrane.

*Nervous symptoms*, such as headache, vertigo, and syncopal attacks, are liable to recur as the anemia and prostration increase and the liability to hemorrhage becomes more frequent. Sudden coma and hemiplegia following upon the rupture of a cerebral vessel (apoplexy) may be the immediate cause of death. Priapism may be very troublesome.



Peripheral paralysis of several cranial nerves, due to hemorrhages into their sheaths, has been reported.

*Cutaneous ecchymoses* are sometimes observed, and sometimes there is a troublesome pruritus. The *urine* contains an excess of uric acid, but albuminuria does not occur, except as a complication.

Along with the anemia and debility are the signs of splenic and lymphatic involvement, and rarely of the bone-marrow. The liver may also become enlarged.

**Leading Symptoms in Detail.**—*The Spleen.*—This organ is generally enlarged in all forms of leukemia, but especially in the spleno-medullary, the most frequent form. It is a prominent feature, both on account of its being the first subject of complaint, and because of the huge size it frequently attains. The enlargement is gradual, and there may be neither pain nor tenderness over it. The tumor may cause a visible projection below the ribs, and in marked cases great abdominal distention may be produced, pushing up the diaphragm and thoracic organs, and extending to the navel in the median line and to the pelvis below. The edge and notch or notches may be felt easily in such instances, while the surface is smooth and the consistence firm. A friction-fremitus is felt sometimes during respiratory movement. The tumor may vary in size, and after severe hemorrhage or diarrhea it may become swollen. Gastric distress after eating and obstructive constipation are usually complained of in cases of great splenic enlargement. Pulsation has been noted and a systolic murmur—"splenic souffle"—has been heard at times over the tumor. The percussion-note is dull.

*Lymphatic Glands.*—In the splenic-lymphatic variety, which is less common than the splenic-myelogenous, and in the still rarer purely lymphatic leukemia, the superficial lymph-glands may be both visibly and palpably enlarged, though not in bunches as in Hodgkin's disease. They are soft, resilient, and movable.

*The Bones.*—Purely myelogenous leukemia is very rare, and local bone-symptoms are scarcely ever manifested. There may be some tenderness on immediate percussion over the sternum or some of the long bones, and slight swelling, irregularity, or deformity of the ribs, the sternum, or other bones may result from leukemic hyperplasia.

*The Blood.*—It is by the blood-examination alone that the pathognomonic features of leukemia are determined. The blood is paler than normal, and sometimes has a brownish-red or chocolate color. Upon a microscopic examination of the blood in the spleno-medullary form of the affection the striking increase in the number of leukocytes is observed at once. The count shows usually from 85,000 to 500,000 white corpuscles per cubic millimeter, and the ratio of the white to the red cells may thus vary from 1 to 150 down to 1 to 10 or 1 to 5 in the average case, instead of the normal, 1 to 500 (see Fig. 38). In extreme cases the number of leukocytes may be equal to, or even slightly greater than, that of the erythrocytes, and such an instance has been recorded by Sørensen, in which the proportion of whites to reds was 3 to 2.

Stained specimens of the blood enable us to recognize the variety of leukemia (see Fig. 39). Thus, in the ordinary *splenic-myelogenous* form the characteristic change is the presence of the abnormal *myelocytes*—large, mononuclear leukocytes with the protoplasm filled with fine neu-



trophilic or occasionally eosinophilic granules. These may make up 25 per cent. of the white cells, whereas they do not occur in normal blood, and very rarely, and only in small numbers, in leukocytosis. They probably correspond to the cells found in the bone-marrow, the large,

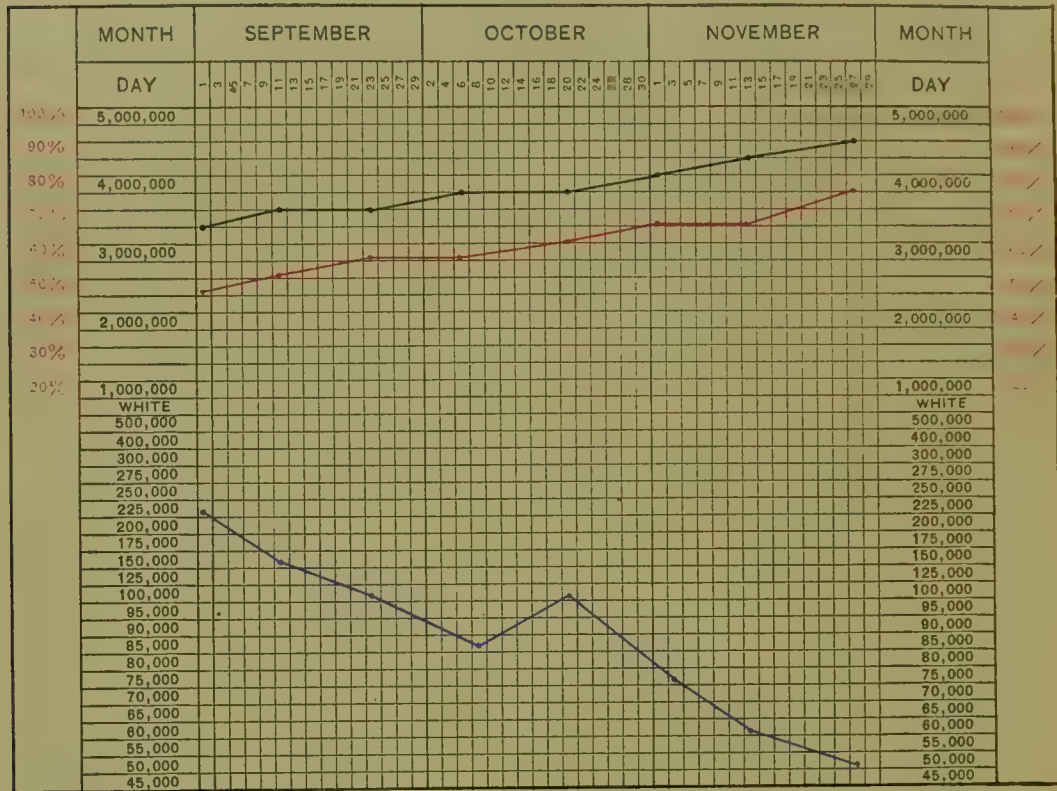


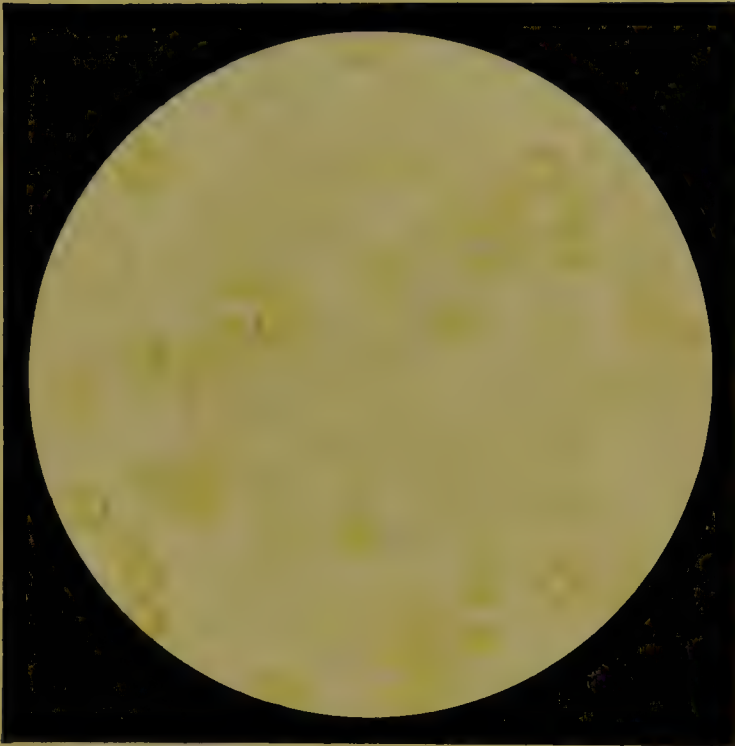
FIG. 38.—Blood-tracing of a case of leukemia. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

oval, and eccentrically-placed nuclei of both blood- and marrow-cells showing karyokinetic figures. The polynuclear leukocytes may be normal in number, but usually they are relatively diminished to about 65 per cent. instead of 75 per cent., as in normal blood. The lymphocytes are also relatively less in number, making up but 1 or 2 per cent., instead of the normal 15–30 per cent. The bright, acid-stained eosinophiles, though absolutely increased, are not always relatively so, though this relative increase may occur. Basophilic leukocytes are rare.

Moderate *oligocythemia* is noted, the reduction being seldom lower than to 2,000,000 per c.mm. The percentage of hemoglobin may also be reduced relatively or in slightly greater proportion. Nucleated red corpuscles, chiefly normoblasts, are frequently found in considerable numbers. Osler asserts that blood of the type of pernicious anemia has subsequently developed a true leukemia. Litten and Musser have also described such cases.

In *lymphatic leukemia*, which is rarer and more quickly fatal than the preceding variety, the blood-changes are also different. The *lymphocytes*—small, mononuclear leukocytes—are the ones increased, all other leukocytes being relatively much diminished in number. Instead of the normal percentage (15 to 30 per cent.), the lymphocytes may number from 90 to 97 per cent. of all the leukocytes. Nucleated red

A



B

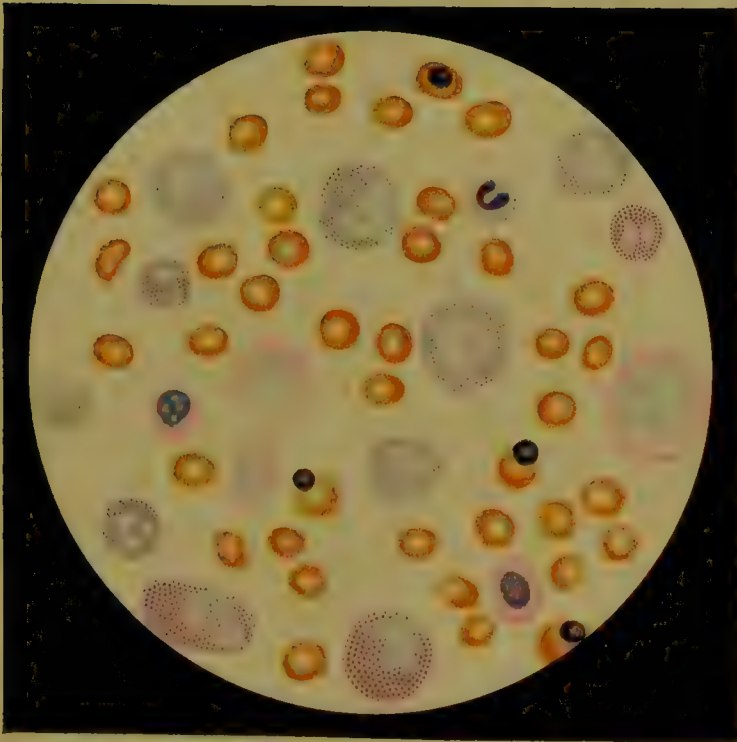


FIG. 39.—A. Fresh preparation from the blood of a case of leukemia ( $\times 550$ ); large mononuclear leukocytes of immature form; B. Preparation of a case of the lieno-myelogenous variety (Ehrlich's triple stain); numerous eosinophilic and immature leukocytes, myelocytes, and nucleated red blood-cells (Grawitz).





corpuscles and myelocytes are absent as a rule, unless the bone-marrow happens to be diseased at the same time. Mixed forms of leukemia are, however, not at all uncommon, so that the proportions of the various types of normal and abnormal cells are quite variable.

The *blood-plates* may be quite abundant in many leukemic cases, and Charcot's octahedral crystals appear in specimens of the blood if allowed to stand for any length of time. An unusually dense and thick fibrous network is also often found.

**Complications.**—Fatal hemorrhages may occur at any time, and pulmonary tuberculosis, pleuritis, pneumonia, septico-pyemia, renal disease, severe diarrhea, and edema may complicate leukemia and cause death.

**Diagnosis.**—This can be made easily and accurately by the blood-examination alone, the distinguishing characteristics of the blood having been enumerated above, both as to the existence of leukemia and the differentiation of its several varieties. It may be necessary in doubtful cases to examine the blood by Ehrlich's staining methods, since the mere excess of leukocytes alone is not proof of leukemia, and also because the disease may exist without an excess, owing either to previous medicinal treatment or to natural temporary improvement.

Leukemia is differentiated from a marked *leukocytosis* by the fact that in the latter there is usually a more moderate increase in the number of leukocytes, and this increase, as a rule, is principally of the polynuclear neutrophiles.

*Hodgkin's disease* may be simulated by the purely lymphatic leukemia on account of the enlarged glands; but in leukemia the lymph-glands are not found in such large bunches, and the blood-examination will show the characteristic changes of lymphatic leukemia if that disease be present. Simply a leukocytosis is present in pseudo-leukemia.

Splenic anemia and lymphatic anemia have not been established as distinct affections, but formerly much time and ingenuity were expended in discriminating them from leukemia and other blood-diseases. Some are cases of pernicious anemia, some of Hodgkin's disease, and some probably are instances of secondary anemia.

Malignant growths of the spleen and lymphatic glands, and also a malarial and passively congested spleen with anemia, may simulate leukemia. The simple leukocytosis here will exclude leukemia.

**Prognosis.**—Many cases are mild and gradual in their progress; children, however, when affected, succumb more rapidly than do adults. Lymphatic leukemia is always fatal earlier than the spleno-medullary variety. Although recovery does occur occasionally, most cases of leukemia, of whatever form, prove fatal certainly within five years, generally in two or three years, and sometimes in seven or eight months or even less (from two weeks to two or more months) in acute leukemia. In an advanced case the prognosis is hopeless. It should be borne in mind that apparent improvement is usually only temporary, and that a fresh exacerbation is apt to follow. Grave symptoms heralding an early termination are profound debility, anemia, emaciation or edema, severe and obstinate hemorrhages, cerebral apoplexy, persistent diarrhea, and high fever. Intercurrent affections not infrequently cause death, while, on the other hand, cases are recorded in which the appearance of intercurrent infectious diseases has favorably affected the course of leukemia.

**Treatment.**—At present no remedies are known to have any permanent curative effect, although several agents are used for their favorable influence. The aim should be to improve the general condition of the patient and endeavor to prolong life by hygienic and medicinal means.

The environment should be made as favorable as possible—physically, mentally, socially, and morally. Out-of-door life in a mild, dry climate, an abundance of nutritious and easily digestible and assimilable food, calm and moderate exercise of mind (depending upon the strength and endurance of the patient), should all be advised and encouraged. On the other hand, traumatism and inflammation, irregular habits of body, worry, excitement, and passionate emotions and appetites should be regulated and avoided.

Arsenic gives the best results in most cases, and should be pushed to the limit of tolerance, as in pernicious anemia. It should be given continuously, regardless of apparent improvement under its use, as the latter may be only the natural remission—a not uncommon incident in the disease. Quinin, iron, and the oil of eucalyptus have been recommended in those leukemic cases in which a clear history of malaria has been obtained. Bone-marrow, either raw and spread upon bread or in the form of a glycerin extract, may be tried when arsenic fails. Oxygen-inhalations and blood-transfusion have been suggested. The so-called “splenic remedies,” whether systemic or local, have no controlling influences upon the disease. Electricity may afford some local comfort or contribute to psychic ease. Complications and intercurrent affections may often be greatly relieved by appropriate treatment.

#### PSEUDO-LEUKEMIA.

(*Hodgkin's Disease; Adenia; General Lymphadenoma; Multiple Malignant Lymphoma; Malignant Lympho-sarcoma.*)

**Definition.**—An anemic disease characterized by the anatomical peculiarities resembling those of lymphatic leukemia—viz. progressive hyperplasia of the lymph-glands, occasional secondary lymphoid growths of other organs (liver, spleen); and by the absence of the destructive blood-changes of true leukemia.

**Varieties.**—Although the disease that bears his name was first described by Hodgkin of Guy's Hospital in 1832 as an affection of the lymphatic glands and spleen, two varieties are included under the title of pseudo-leukemia (or Hodgkin's disease), as follows: (1) that which presents simply an enlarged spleen (the less frequent one); and (2) that in which the lymphatic glands are chiefly involved.

**Pathology.**—The lymph-glands show different degrees of hyperplastic enlargement and consistency. In the earlier stages they are small, isolated, and movable, while in advanced and well-developed cases of the disease they are larger, fused together into great bunches, and more or less fixed by fibrous investment. As a rule, the glands are soft and elastic, though sometimes they are hard and dense, and masses as large as an orange or pineapple may be seen. Single glands may be as large as a hen's egg, and the gland-capsules may show connective-tissue proliferation and a thickening periadenitis. Extension of the lymphatic



growth into the surrounding tissues by perforation of the capsule may occur. As a rule, the overlying skin is freely movable, though it may rarely be adherent. On section the tumors display a smooth white or reddish-gray surface in the case of the soft and almost fluctuating glands, and a grayish or a yellowish-white color if they are firm. The fusion of the swollen glands into nodular masses is also seen, and when ulceration through the skin has taken place suppuration of the glands may be revealed. In the harder tumors areas of necrosis having the appearance of caseation may be visible, and shining, more or less hyaline masses of fibroid tissue may also be detected.

Microscopically, there is a typical hyperplasia of the lymph-cells, often obscuring completely the reticulum of the gland, except in the harder enlargements, where the fibrous proliferation shows a very distinct network. The arrangement of the lymph-tracts is distorted and disturbed in the larger growths only.

The cervical glands are most prominently involved. The superficial chains of glands—axillary, mediastinal, scapular, and pectoral—especially along the great vessels, are often found connected, and the inguinal, bronchial, and lumbar glands are also affected, though less frequently. The retroperitoneal glands are more frequently affected than the mesenteric, and sometimes the thoracic vessels are completely surrounded by enlarged lymph-glands; they have occasionally projected externally by perforation through the sternum. The abdominal vessels, nerves and nerve-plexuses, and ducts may be compressed also by huge groups of enlarged glands.

The *spleen* is enlarged in about four-fifths of the cases, but only slightly. In the majority of cases there are disseminated throughout the organ whitish, lymphomatous growths or nodules from the size of a pea to that of a nut. Their histological structure is like that of the lymph-glands. Occasionally the spleen alone is hyperplastic.

Lymphomata may also develop in the tonsils, lingual follicles, intestinal lymphatics, liver, kidneys, lungs, brain, heart, testicles, retina, and skin. Invasion of the spinal cord may occur by erosion of the vertebræ or through the blood-current by metastasis. The bone-marrow often has the same appearance as in pernicious anemia.

**Etiology.**—There are no well-established predisposing conditions to which Hodgkin's disease is referable. In the larger number of cases males are affected, and young and middle-aged persons—between the age, of ten and forty years—seem to be attacked in about 70 per cent. of the cases. Heredity may possibly be a cause. Neither has an exciting cause been discovered as yet. The disease would seem to belong to the group of infectious granulomata, but the infectious agent is not known. Flexner thinks that certain protoplasmic foreign bodies (found in the larger nodules of two cases) may possibly have a causal relation to the disease. Malaria, syphilis, chronic skin-diseases, and various irritative conditions, especially of the mouth, giving rise to local glandular swellings, have also been assigned as causes. In undoubted instances of Hodgkin's disease the lymphatic glands frequently harbor tubercle bacilli; hence it has been thought that the latter exercise a distinct causative influence. It must be remembered, however, that some of these may be examples of secondary accidental infection; others of



primary diffuse lymphatic tuberculosis, indistinguishable from or mistaken for Hodgkin's disease. It is not uncommon to find pseudo-leukemia developing in a person who immediately preceding the beginning of the disease was apparently in perfect health.

**Symptoms.**—Usually the first thing to attract attention is the enlargement of the submaxillary and cervical glands, often on one side of the neck alone. These grow gradually until they may finally appear on both sides as large as a fist, and produce considerable disfigurement. Sometimes several years may elapse before other glandular groups are affected, but, as a rule, it is a matter of months only before the axillary, then the inguinal, and perhaps the internal, glands are invaded. The changes vary greatly in rapidity and extent.

At first the general health may be but slightly affected. A little constitutional disturbance and some pallor may be complained of, though seldom before the glandular swellings are noticed. Then as the disease progresses the paleness increases and all the symptoms of a marked anemia appear—languor, failure of physical strength, beginning emaciation, gastro-intestinal derangement, headache, giddiness, palpitation, dyspnea, and edema of the legs. Later, the serous cavities contain

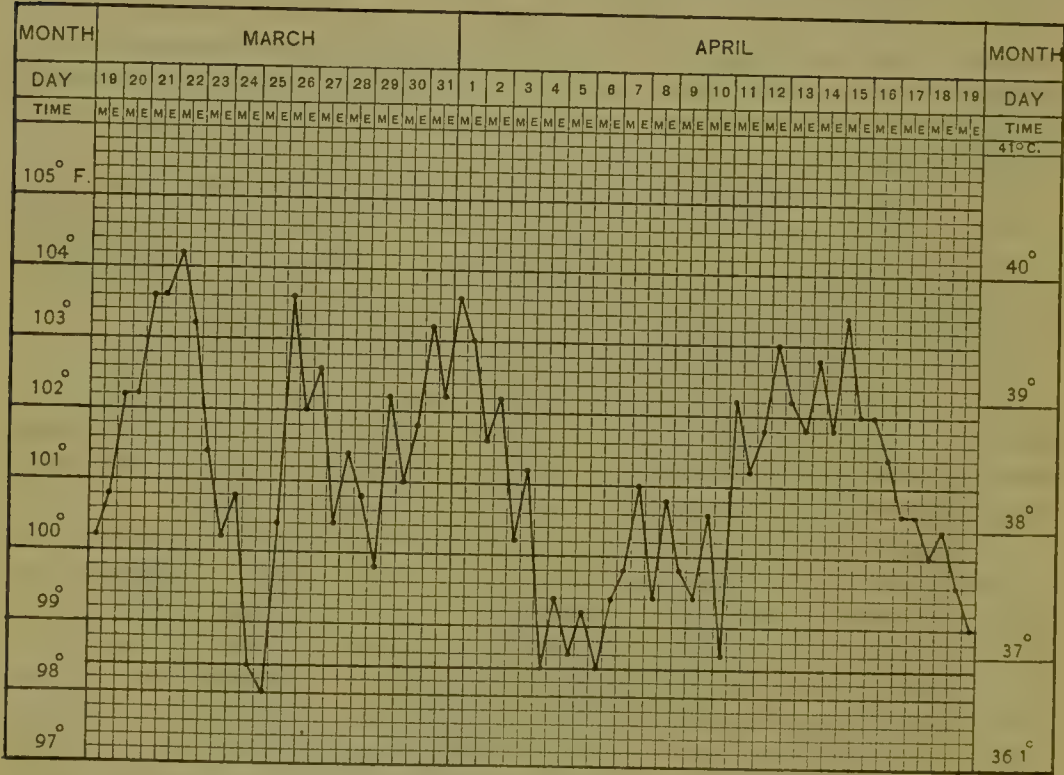


FIG. 40.—Temperature-chart of a case of pseudo-leukemia.

effusion and there is a tendency to hemorrhages. Epistaxis and metrorrhagia are apt to occur, and petechial spots, especially on the lower extremities, are not infrequent. The physical signs of anemia—hemic murmurs—are also present. An irregular slight or moderate pyrexia is common to most cases. Fever of a peculiar intermittent type has been observed, the intermissions and paroxysms each lasting for several days or weeks (see Fig. 40), and the term “chronic relapsing fever”

has been applied in consequence. When these pyrexial exacerbations occur the cases generally run a more acute course.

The symptoms due to mechanical compression by the lymphomata are varied and numerous, depending upon the number, size, and distribution of the tumors. Hundreds of tumors may be present throughout the body, but, unless they press upon the adjacent nerves, the glands are not usually painful. Enlargement of the tracheal and bronchial glands may cause dysphagia, dyspnea, thoracic pain, disturbed phonation, and venous congestion, by pressure respectively upon the esophagus, trachea, bronchi, thoracic nerves, recurrent laryngeal nerves, superior vena cava, and the jugular veins. The obstruction to respiration may become so great as to produce death by suffocation. *Congestion* of the head and upper extremities may be quite marked, and in such cases compensatory dilatation of the superficial veins is observed. *Edema* of the hand and arm may result from venous obstruction due to the pressure of very large axillary glands. *Deafness* may be produced by growths within the pharynx. The *heart's action* may be disturbed by pressure on the pneumogastric, and the heart itself may be dislocated by great gland-tumors within the chest. Under such circumstances the latter may be detected by dulness on percussion over the anterior mediastinal space. Inequality of the pupils and unilateral sweating of the face, owing to glandular pressure upon the cervical sympathetic, may be noticed in some cases.

*Edema* of the feet and legs may be an early indication of enlarged abdominal glands pressing upon the femoral veins. Sharp lancinating pains along the nerves may also be felt. *Jaundice* is sometimes attributed to pressure upon the bile-duct. *Gastro-intestinal disturbances* may be troublesome, and are usually symptomatic of lymphoid growths in the stomach and bowels. In thin individuals gland-masses may be palpable over the abdomen. It has been suggested that the bronzing of the skin sometimes seen in Hodgkin's disease may be due to the pressure of enlarged glands upon the suprarenal capsules. The slightly or moderately enlarged spleen can usually be felt just below the ribs, projecting toward the navel. *Pressure-paraplegia* and *neuralgic pains* variously distributed throughout the body should also be mentioned among the nervous manifestations. Tenderness over the spleen and bones may be elicited. An intense pruritus has been complained of, and the skin may be erythematous. Albuminuria is not uncommon; ascites and hydrothorax are late conditions, and occasionally the thyroid and thymus glands are involved. The characteristic feature in splenic pseudo-leukemia is the decided enlargement of the spleen without involvement of the lymphatics.

The *blood* shows a moderate diminution in the number of red corpuscles, and a corresponding diminution in the hemoglobin, the former in most instances numbering from 2,000,000 to 4,000,000 per cubic millimeter. There may be more or less leukocytosis, and sometimes the lymphocytes may preponderate relatively; if the latter be present in great numbers, the blood may show great similarity to that of lymphatic leukemia. An occasional normoblast may be seen.

**Diagnosis.**—Pseudo-leukemia is more readily confused with *tuberculous adenitis* than any other disease, particularly at the outset. Although



an acute tuberculous adenitis may very closely simulate Hodgkin's disease and render a diagnosis almost impossible, more often the glands of tuberculous adenitis are slower in enlarging and extending than in this disease. In fact, extension of the lymphatic enlargements of tuberculosis is rarely seen as compared with pseudo-leukemia. Again, tuberculous adenitis is most common in the young, is unilateral rather than circumferential in the neck, and attacks the submaxillary glands oftener than the cervical chains along the sterno-cleido-mastoid. Again, periadenitis, adhesion, and suppuration of the glands occur in tuberculosis. Tuberculous foci in other organs may also be found. Intermittent attacks of pyrexia are an indication favoring Hodgkin's disease. In doubtful cases a gland may be removed for microscopic examination.

The blood should be examined in order to differentiate from *leukemia*.

*Syphilis* must be carefully excluded by the history, symptoms, and therapeutic test. *Neoplasms* of the lymph-glands may sometimes be difficult to distinguish from pseudo-leukemia.

**Prognosis.**—This affection runs an almost invariably fatal course. The variability of the symptoms, of the rate of growth, size, and extension of the lymphatic enlargements, and the remissions and exacerbations of the disease are, however, notable. In some cases the termination may occur in a few months, but usually death ensues after the lapse of two or three years. Recovery is not impossible in the early stages of the disease, but in fully-developed cases the prognosis is absolutely unfavorable. It should be remembered that some instances of Hodgkin's disease seem to merge into a true lymphatic leukemia.

Grave indications are the rapid extension of the glandular enlargements, great debility, anemia, emaciation, steadily increasing and continuous pyrexia, thoracic pressure-symptoms, hemorrhages, and marked anasarca. Sometimes the tumors diminish greatly before death. In certain cases general streptococcus infection, hydrothorax, edema of the lungs, mechanical compression, hemorrhage, intercurrent diseases, or such complications as empyema or nephritis, often preceded by coma, may be the immediate cause of death.

**Treatment.**—Local or surgical treatment is of no avail. Hygienic measures and the use of all possible agencies to support the strength of the patient should be resorted to, and the administration of arsenic in gradually ascending doses, as for pernicious anemia and leukemia, should be begun as soon as the diagnosis of pseudo-leukemia is made. The value of arsenic is undoubted in many cases, and Fowler's solution is very generally used. Phosphorus has also been recommended. Inunctions of ichthyol, iodoform, or green soap may be tried for their psychic effect, and the galvanic current may also be applied topically. Tonics and nutrients may be of temporary service.

#### ANÆMIA INFANTUM PSEUDO-LEUKÆMICA.

**Definition.**—The above title was given by von Jaksch to a form of anemia occurring in childhood that bears certain similarities to leukemia, but is without the tendency to a fatal end. It is probably the same class of cases that Italian writers have classified under the name of *anæmia splenica infettiva dei bambini*.



**Pathology.**—Splenic enlargement is the most striking lesion. The organ is hard and dark red, and perisplenitis may be observed. The histological examination shows a uniform hyperplasia of the tissue, such as is witnessed in ordinary splenic hypertrophy due to various conditions. The liver is enlarged in most cases, but presents practically normal appearances on section; slight enlargement of the lymphatic glands may also occur, though never lymphomatous tumors. Diffuse reddening of marrow has been described.

**Etiology.**—Children under the age of four, and particularly during the second half year of life, are especially prone to this condition. It is equally common in the two sexes, and is most often met with in rachitic infants, 16 of 20 cases collected by Monti and Berggrün having exhibited this etiological factor. Hereditary syphilis, intestinal disturbances, and other diseases doubtless play a part in the etiology. The disease is a rare affection, occurring very seldom even in the largest children's clinics.

**Symptoms.**—The onset is gradual. The child becomes pale, weak, and often emaciated, and enlargement of the spleen is the most striking feature. Sometimes this reaches such a grade that the left half of the abdomen is practically filled, variations in its size being observed from time to time. Hepatic enlargement is frequently present, but does not correspond to that of the spleen, and the lower border of the organ is found to be sharp instead of rounded, as is the case in leukemia. Gastro-intestinal disturbances may occur in the course of the disease, and gradually increasing weakness may lead to a fatal end; death may also occur from peritonitis, bronchitis, or pneumonia.

**Blood.**—An examination of the blood will in many cases show an inordinate reduction in the number of red corpuscles. Nearly always the number is below 3,000,000. Degeneration of the corpuscles, polychromatophilia, and poikilocytosis are seen in the severer cases. Large numbers of nucleated erythrocytes, especially the normoblasts, may be found, and karyokinetic figures are frequently observed in the nuclei. A marked increase in the number of leukocytes is one of the characteristics, the number ranging from 40,000 to over 100,000, and the proportion of the red to the white at times being as low as 12 to 1. Considerable fluctuations in the number of leukocytes may occur from time to time. Von Jaksch insisted that the different forms of leukocytes occur in their usual relative proportions, or that the polymorphous forms are specially increased. Cases have, however, been described in which the mononuclear elements were particularly increased.

The *nature of the disease* is difficult to determine, though the favorable termination of many cases, the lesser grade of hepatic enlargement, and the character of the leukocytosis distinguish these cases from leukemia. It is not even certain that it is a special disease-entity, and the evidence is in favor of its being a type of secondary anemia with peculiar features, due perhaps to the constitutional condition and the age of the patient.

**Diagnosis.**—Some points of distinction from leukemia have been referred to above. In addition I would say that the absence of hemorrhages, purpura, and lymphomatous enlargements, and the presence of abundant nucleated corpuscles showing karyokinesis, together with the existence of rickets, point to a non-leukemic affection.

**Prognosis.**—The disease tends to a progressive increase of the anemia, but under treatment the majority of cases terminate favorably.

**Treatment.**—Hygienic measures together with the administration of remedies directed to the anemia constitute the treatment.

#### CHLOROMA.

Owing to its clinical resemblance to leukemia and pseudo-leukemia a brief reference to this comparatively new and rare affection may be made here. Pathologically, it consists of a sarcomatous growth, the primary seat of which is in the periosteum and bone in and about the orbit. The growth shows a pea-green pigmentation. Secondary growths may be widespread, the metastatic nodules being also green in color, but more circumscribed than are the lymphatic infiltrations of ordinary leukemia. In 2 cases reported recently, 1 by Dock and the other by Ayers,<sup>1</sup> the ages were fifteen and seven years respectively.

Pain in the orbital region, exophthalmos, and deafness were noted early, and severe conjunctival hemorrhages and epistaxis occurred. Rounded elastic swellings were observed in the temporal and parotid, as well as in the orbital regions, corresponding to the chloromata. The blood was pale and watery, and leukocytosis was present, multinuclear leukocytes being noted. The eosinophiles were slightly reduced.

The course of the disease—spoken of by French writers as “green cancer”—is rapid, and death usually comes on within a few months.

### DISEASES OF THE DUCTLESS GLANDS.

#### DISEASE OF THE SUPRARENAL CAPSULES.

##### ADDISON'S DISEASE.

**Definition.**—A constitutional disease, characterized by a degeneration of the suprarenal capsules or semilunar ganglia, a bronzed or pigmented skin, great bodily and mental asthenia, feeble circulation, and gastro-intestinal irritability.

This affection is named in honor of its discoverer, Thomas Addison of Guy's Hospital, London, who first described it in a monograph published in 1855, entitled “The Constitutional and Local Effects of Disease of the Suprarenal Capsules.

**Pathology.**—Addison emphasized the fact that while the suprarenal bodies were affected with a fibro-caseous alteration in many cases, the anatomical changes were by no means always the same. Both suprarenal capsules are usually diseased at the same time. Tuberculosis is the commonest condition, and is often associated with tuberculous lesions in other parts of the body, as in the lungs, bones, and other glands. Rarely, it seems to be primary, no other evidences of tuberculous infiltration being found. The capsules are enlarged, firm in places, and nodu-

<sup>1</sup> *Jour. Amer. Med. Assoc.*, Nov. 7, 1896.

lated on the surface, owing to the caseous masses surrounded by fibrous tissue. Sometimes there is marked cicatricial contraction of the adrenals, and the adjacent structures may be found matted together with the capsules. Microscopical examination shows a reticulum of connective tissue surrounding a soft cheesy, granular, and fatty detritus, lymphoid cells, and some giant cells. Other morbid processes in the adrenals that are non-tuberculous in nature have also been found associated with Addison's disease, such as atrophy of one or both glands from interstitial cirrhosis, carcinoma or sarcoma, and chronic inflammation.

Especial attention has recently been given to the condition of the *solar plexus* and *semilunar ganglia* of the abdominal sympathetic, and implication of these nervous structures by compression, cicatricial contraction, or by chronic inflammation, is not infrequently discovered, together with a degeneration of the nerve-cells.

Enlargement of the solitary and agminated follicles of the intestine, and slight enlargement and some softening of the spleen are noted at times; parenchymatous or fatty degeneration of the heart, liver, and kidneys has also been noted in some instances. The thymus gland may be found to have remained normal, or even to have enlarged, perhaps. The deposition of pigment is in the same anatomical elements as in the negro—in the lower layers of the rete Malpighii.

The pathological connection between the symptomatic phenomena of Addison's disease and the anatomical lesions has not been satisfactorily made out. The experimental evidence regarding the functions of the adrenals is imperfect; but it seems quite probable that some essential "internal secretion," influencing the normal metabolism of the skin and muscles, is diminished or absent in Addison's disease. On the contrary, cases exhibiting the clinical phenomena of this affection have occurred in which no suprarenal morbid processes could be found *post mortem*. Again, marked changes have been observed in these glands, while during life no symptoms of the disease had been noted. Hence, it is maintained by some that the abdominal sympathetic nerves and ganglia are directly concerned in producing the clinical manifestations, either by an independent morbid process or by extension from some adjacent organ. Others hold that when both the adrenals and sympathetic ganglia are the seat of pathological changes, the latter directly and the former indirectly lead to Addison's disease. The data are not sufficient, however, to determine whether the principal involvement is nervous or secretory, and to future investigations must be left the decision in regard to this point.

**Etiology.**—This is obscure. It has been held that some infection of the blood from without precedes the suprarenal and nervous lesions of Addison's disease. A *tuberculous* diathesis or infection has also been emphasized by some investigators, and a history of *injury* to the trunk has been noted in several cases. The disease is more common in Europe than in America, though it is rare everywhere. Sixty per cent. of the cases occur in males, and while the disease may affect all ages (it may even be congenital) it is usually found in early or middle life—between fifteen and forty years of age. That Addison's disease is due either to a general neurosis or to disturbed hematopoiesis is merely hypothetical.



**Symptoms.**—While it does not seldom happen that tuberculosis or carcinoma affects the adrenals, the purest and most typical symptoms of Addison's disease are apparently primary in their development, and not those that usually attend the course of the former diseases.

The gradual pigmentation of the skin of various parts of the body may be one of the first evidences of the affection. This pigmentation may have either a dusky-yellow, bronze or yellowish-brown, olive, deep or greenish-brown, or even black color. Although sometimes diffuse, the discoloration is not uniform over all parts of the body, but commences earlier and becomes deeper especially on the exposed parts and where the normal pigmentation is marked, as the face, neck, backs of the hands, the axillæ, abdomen, groins, genital regions, and the areolæ of the nipples. Pigment-spots, often somewhat bluish in color, are also found on the mucous membranes of the mouth, lips, conjunctiva, and vagina. On the lips the discoloration takes the form of a dark streak, running lengthwise, near the junction of the skin and mucous membrane; or brownish patches or streaks corresponding to points of pressure by the teeth may be noticed. Irregular stains with ill-defined borders may also be shown on the skin, corresponding to the lines of pressure exerted by garments, strings, suspenders, garters, etc. (Greenhow). White patches of leukoderma may be seen here and there in marked contrast to the pigment-deposits.

The constitutional symptoms may exist in a slight degree before the pigmentation first attracts the patient's attention. There is gradual and progressive *asthenia* without apparent cause, great lassitude and loss of physical and mental energy, breathlessness, palpitation, headache, dizziness, tinnitus aurium, sighing and fatigue, and the functional murmurs that are observed in the anemias. The *blood-examination*, however, rarely shows any marked reduction of the erythrocytes or hemoglobin; nor is there any leukocytosis. The *heart's action* is weak and the pulse small and feeble; attacks of faintness on exertion are common, and coldness and clamminess of the extremities are often complained of. The *general nutrition* may suffer considerably, though often the fat, particularly of the abdomen, is well preserved.

*Gastro-intestinal symptoms* are usually prominent. There is a loss of appetite, and nausea and vomiting may occur early and either be paroxysmal or persistent. The tongue may be clean, and the gastric disturbances do not seem to follow errors in diet. Diarrhea may be troublesome in the latter stage, and is often associated with intractable vomiting. *Neuralgic attacks* of either sharp or dull, aching pain are referred to the epigastric, hypochondriac, and lumbar regions in about one-third of the cases. The mind is usually clear until near the last, but mental weariness is constant, and, as the later stages of the disease come on, the patient often lies in a somnolent, semi-comatose state. The physiognomy expresses fatigue, dejection, and apathy; the speech becomes slow and incoherent, and in many cases the patient passes into delirium. Prostration is profound, the weakness being disproportionate to the general condition.

Polyuria is sometimes evident, but albumin is seldom present. The amount of indican is increased, as it is in the urine of all of the cachectic diseases associated with destruction of albuminoids. There

is usually a diminished excretion of urea, but urobilin and uromelanin may be present in abnormal quantity. Tubercle bacilli may be found in the sputum.

**Diagnosis.**—The principal error in diagnosis is in the assumption that the case is one of Addison's disease, simply from the presence of patches of pigmented skin. Other conditions in which the discoloration may simulate that of Addison's disease are the following: (1) Carcinomatous and tuberculous disease, particularly when seated in the abdomen and when involving the peritoneum; (2) Hepatic disease, such as the cirrhosis of diabetes, protracted jaundice, chronic congestion, and lithemia ("liver-spots"); (3) Pregnancy, and uterine disease, in which the patchy discolorations (chloasmata) appear principally upon the face; (4) Irritation of lice and dirt and exposure, as in the case of tramps and vagrants ("vagabond's disease"); (5) Tinea versicolor; (6) Melanotic sarcoma; (7) Exophthalmic goiter; (8) Post-eruptive staining of syphilitic eruptions; (9) The administration of silver nitrate for a long time (argyria); (10) Marked brunette complexions and racial admixture.

When the pigmentation is scanty, of course the diagnosis is more difficult; but in all cases of pigmentation in which other causes may be excluded the progressive asthenia, unaccountable vomiting and diarrhea, easily compressible pulse, great bodily weakness, mental hebetude, and lumbar and epigastric pain render the diagnosis of morbus Addisonii, or *melasma suprarenale*, justifiable. It is to be remembered that the bronzing of the skin may precede as well as follow the constitutional symptoms.

In the negro the diagnosis of this affection is extremely difficult, both on account of the naturally dark skin and because of the dark discolorations of the oral mucous membrane, found even in health in many individuals.

**Prognosis.**—The course of Addison's disease is almost always chronic, though cases have been reported occasionally in which the onset has been sudden, with febrile phenomena and a comparatively acute course of a few months, or weeks even. Usually the disease lasts about one year, although some cases may continue over five or even ten years. Temporary remissions may be observed, but death is inevitable in by far the majority of instances. The termination is gradual, and by profound asthenia, or sometimes by coma, delirium, or convulsions (epileptiform).

**Treatment.**—The hygienic and medicinal treatment must have virtually the same objects in view as in the other grave cachectic diseases, and is both sustentative and symptomatic. As quiet a life as possible should be strictly enjoined, owing to the dangers of a sudden and fatal syncopeal attack. Rest in bed is necessary in moderate and advanced cases during a part of the day for the former and constantly for the latter. The diet should be restricted to light nutritive, concentrated, and easily assimilable food, and particularly to the nitrogenous or proteid substances. An absolute milk diet may be necessary in some cases.

Iron and arsenic may be administered in the anemic cases, and strychnin, guaiacol carbonate, phosphorus, and the nuclein preparations may also be given, along with bitter tonics. Bismuth and salol may be



of great service in controlling the diarrhea that often occurs. The nausea and vomiting may be relieved by unfermented grape-juice, albumin-water, champagne, cracked ice, cerium oxalate, creasote, and the like. Electricity is often a valuable adjunct in the treatment of the muscular weakness and nervous exhaustion, and even in reducing the pigmentation. Loss of sexual desire and power were also restored in a case reported by Flint.

It seems quite probable that the administration of the extract of suprarenal capsules will prove to be of considerable value in causing marked improvement, if not a permanent cure, in a certain percentage of cases. The therapeutic efficiency of this more or less physiological remedy is still to be proved, but several cases have been reported in which distinctly good results have been obtained. In one instance mentioned by Osler, in which a glycerin extract of a pig's suprarenal was given at first in doses of half a glass three times a day, improvement was noted in the temperature, pulse, weight, and physical and mental vigor from the first week of the treatment, which was continued for three months and a half. Eight months after the treatment was begun the patient appeared to be well and strong, and attended to business; the pigmentation, however, was not removed. In a recent case of my own this remedy produced like results. For the present, however, too positive a value should not be attributed to the suprarenal extract, owing to the meager data at hand.

## DISEASES OF THE THYROID GLAND.

### THYROIDITIS.

**Definition.**—Acute inflammation of the thyroid gland. The gland may either have been previously healthy or the seat of a goitrous enlargement; when inflammation attacks previously diseased or enlarged thyroid tissue the term *strumitis* is often used.

**Pathology.**—The gland is swollen, boggy, and the seat either of a single large or of multiple small abscesses; the numerous large blood-vessels are engorged; and hemorrhages, thrombi, and areas of tissue-necrosis are frequently found. Sometimes evidences are seen of the burrowing of the abscess around the trachea and esophagus, and erosion of the laryngeal cartilages and perforation of the respiratory and digestive tubes have also been noted. Gangrene of the cervical tissues may follow an intense inflammation.

**Etiology.**—Thyroiditis is seldom primary in origin. It may be caused by traumatism, but usually it is secondary to one of the infectious diseases, such as small-pox, typhus, typhoid fever, or malaria. Rheumatism has also been given as a cause. Hemorrhages into the substance of a goiter, whether apoplectic or traumatic, may predispose to a strumitis that may be excited by the introduction of streptococci by an unclean needle, etc. Repeated congestions of the thyroid or a simple acute congestion may also dispose to thyroiditis. Thus, abrupt suppression of the menses and sexual excitement may cause an acute goiter (congestion); the gland is also found engorged in many pregnant women, and is at such times more susceptible to inflammations.

**Symptoms.**—There are fever, pain, swelling, and suppuration in



one or the other lobe of the gland. Venous obstruction may be serious and give rise to vertigo, headache, cyanosis, and epistaxis; and compression of the windpipe by the great swelling may cause death before the abscess bursts. Resolution occurs infrequently, especially in the "strumous" cases. Indeed, the symptoms of a strumitis are usually more severe, owing to the greater size of the thyroid, a tendency to metastasis, and to the burrowing of pus into adjacent tissues leading to perforation and rupture of the abscess into the trachea or esophagus.

**Diagnosis.**—Thyroiditis must be differentiated from the *laryngeal perichondritis* that is also seen in the course of infectious diseases, as typhoid fever and small-pox. The higher and more median position and the smaller swelling of laryngo-chondritis are distinctive points.

**Prognosis.**—The outcome is usually favorable in all cases in which spontaneous rupture occurs externally or when evacuation of the pus is effected. Strumitis runs a less favorable course for the reasons mentioned above, and from the fact that the constitutional vitality in such cases is less resistant and the probability of cure is to that degree diminished. Extension of the suppuration into the deeper tissues of the neck is of grave import.

**Treatment.**—This is antiphlogistic and surgical. The pus must be evacuated freely, and sometimes tracheotomy or thyroidectomy may be necessary in order to save life.

#### GOITER.

(*Bronchocèle.*)

**Definition.**—A chronic hypertrophy and hyperplasia of a portion or the whole of the thyroid gland. It is of obscure origin, involving one or more of the structural tissues, and is subject to various degenerative changes.

**Pathology.**—Several different varieties are described. In the *simple hypertrophic* or *parenchymatous* form there is a hyperplasia of all the original tissue-elements. The *follicular* form shows an increase of the true glandular elements alone.

*Fibrous goiter* is that variety in which the interstitial tissue or stroma is increased out of all proportion to the hyperplasia of the follicles, which are also involved in a much slighter degree. This variety of goiter may have an inflammatory origin (thyroiditis). In old cases marked sclerosis may be assumed. There is also a *vascular* variety, in which the blood-vessels are enormously dilated. More commonly the veins are affected; but in the so-called aneurysmal variety the arteries are chiefly involved. The intense venous variety of vascular goiter has been designated "cancerous tumor of the thyroid," and the whole gland may in such cases be quite elastic and like spongy erectile tissue. Follicular hyperplasia is often associated with vascular enlargement.

The special varieties of goiter due to degenerative changes are the *cystic*, *amyloid*, *colloid*, and *calcareous*, and of these the first named is the most common. It consists in the development in a large goiter of one or more large or small cysts filled with different kinds of fluid of varying consistency. Sometimes the liquid is colloid or mucinous in nature, and it may be chocolate-colored and contain the residue of hemor-

rhages (cholesterin, fatty products, and albumin). Amyloid changes affect principally the vessels; colloid changes are also frequent, while calcareous infiltration is seen in very old fibrous goiters. Inflammation and suppuration of the goitrous gland may ensue.

**Etiology.**—Goiter may occur anywhere sporadically. Endemically and in its worst forms it occurs in the mountainous districts of Europe, Asia, Mexico, and South America, particularly in the Alps, Pyrenees, and Andes. It has also appeared in certain limestone regions, such as New England and Ontario, Canada, where the *habitual use of limestone-water* for drinking purposes seems to induce the disease. *Heredity* undoubtedly plays a part in its causation, certain children having been born with goiter. Occasionally it has become epidemic in certain sections of the goitrous districts in Europe where military garrisons have been stationed, thus indicating the possibility of some infectious influence. Women are more liable to goiter than men, and it is more common to find it after ten or twenty years of age. It has been alleged that pregnancy also influences the development of this condition.

**Symptoms.**—The enlarged thyroid is readily recognized and felt, though the patient may complain of nothing but the disfigurement, except when the tumor is of sufficient size to cause symptoms of compression. The goiter develops very gradually, and may vary in dimensions from the merest perceptible enlargement to a growth that overhangs the chest and greatly hinders the movements of the head. It may or may not be uniform in its development, and is often more enlarged on the right side and in front than on the left side. It is not infrequently observed to increase in size with each succeeding pregnancy and during or after each menstrual flux.

The tumor is painless, is not adherent to the overlying skin or to any of the neighboring bones, and rises and falls during the act of swallowing, moving with the larynx. The veins covering it are swollen and prominent. It interferes with respiration oftener than with deglutition, causing dyspnea; alteration or loss of the voice may also ensue. Displacement and distortion of the trachea, the vessels, and other cervical tissues may be produced. Large pendulous growths usually cause less serious discomfort than the small encircling tumors that extend downward into the thorax. Headache, somnolence, and marked cerebral symptoms, such as tetany and convulsions, have been described as due to compression of the carotids.

The general health or nutrition seldom fails unless inflammation and suppuration (strumitis) attack the goiter during the course of some infectious disease, as not infrequently happens, or in cases in which the thyroid function is abolished, leading to the profound nutritional and cerebral disorders of cretinism in children or myxedema in adults.

Dettrich and Osler have each reported an instance of a goitrous growth affecting aberrant portions of thyroid found in the upper region of the pleural cavity, one on the right and one on the left side.

Sudden death may ensue in a few cases, either from pressure on the vagi, from a severe intraglandular hemorrhage, or from a hemorrhage into the adjacent cellular tissues.

Auscultation often reveals a loud blowing murmur, especially marked in the vascular bronchoceles. Palpation over the tumor often shows



the bossellated surface present in cystic goiter; fluctuation may also be detected in such cases, as well as over the abscess of a strumitis.

**Diagnosis.**—Goiter is easily differentiated from other enlargements. The constant location and the character and course of growth of the bronchocele are distinctive. If both lobes of the thyroid are affected, making a symmetric swelling, the diagnosis is almost assured. Bronchocele is not easily confounded with other cervical tumors, such as lymphadenoma, glandular tuberculosis, carcinoma or abscess of the thyroid, or sebaceous cysts. A characteristic feature of tumors of the thyroid is their vertical movement during the act of deglutition.

**Prognosis.**—This is guardedly favorable as to life, but unfavorable as to cure. The course is chronic, but the possibility of a sudden fatal termination should be borne in mind.

**Treatment.**—Prophylaxis should be practised in goitrous districts by the drinking of boiled water only, and removal to a non-goitrous region is advisable. The majority of drugs recommended for internal and external use have been proved valueless, though in the parenchymatous and follicular forms potassium iodid by the mouth and the vigorous and methodic use of iodin over the tumor have been much lauded. Mercurial ointment—the red or biniodid especially—has also been recommended for local application. Ergot or belladonna in progressively increasing doses may do good in vascular goiters. The younger and softer goiters may also be benefited by electrolysis, needles attached to the negative pole being inserted into the substance of the tumor while a large sponge or clay positive electrode is placed in the vicinity.

In the older, fibrous, and degenerated goiters surgical treatment alone may be of service. Injections of iodin, tapping of cysts, incisions of the isthmus, and ligature of the thyroid arteries have been practised among the lesser operations. Thyroidectomy, or a partial extirpation of the thyroid, is the radical and final operation.

Recently, the fresh, chopped thymus gland of the sheep, spread on bread, was given in 20 cases of follicular and parenchymatous goiter with gratifying results. The pressure-symptoms were relieved and a perceptible diminution in the size of the goiter was demonstrated by actual measurements. Complete recovery, in an anatomical sense, however, was realized in two cases only.

#### EXOPHTHALMIC GOITER.

(*Graves's Disease; Basedow's Disease.*)

**Definition and Nature.**—Although the view cannot be unreservedly accepted, exophthalmic goiter is probably of thyroid origin and is dependent upon an abnormal action (or over-action) of the thyroid gland; it is characterized clinically by tachycardia, tremors, enlarged thyroid, and exophthalmos. Among other leading theories the following may be briefly stated: (1) that it is due to disturbed innervation (Buschan); (2) that the seat of the disease resides in the medulla oblongata; (3) that it is an affection of the sympathetic nerves; and (4) that it is a disease of the central nervous system associated with a chronic intoxication.

The theory held by Möbius, that exophthalmic goiter is attributable primarily to a disturbance of the function of the thyroid ("hyperthyroid-



ation"), a condition directly opposed to the lack of thyroid function, as in myxedema, is amply supported by clinical evidence, the complex symptom-group of the former being directly antagonistic to that of the latter disease. Thyroid-feeding, moreover, while it sometimes causes parenchymatous goiters to disappear rapidly, usually aggravates the symptoms of Basedow's disease. Regarding the *pathologic changes* in the thyroid little is known. Brissaud<sup>1</sup> found in 25 cases of various chronic diseases changes in the thyroid, and, although the glands in exophthalmic goiter showed no changes peculiar to that disease, yet quantitatively the lesions were always such as to make "hyperthyroidation" possible.

**Etiology.**—It is more common in women than men, and, although it has been met with at both extremes of life, it is seen usually in adults. The influence of heredity is undoubted, and several members of a family may suffer, persons that possess a sensitive nervous organization being especially prone to the disease.

Among direct causes are emotional disturbance, worry, severe acute disease (noted in a recent case of my own), and prolonged mental or physical strain.

The disease may also occur as a secondary complication in the course of simple goiter, affections of the nose, and pregnancy; this variety, however, is to be distinguished from the primary or essential form.

**Symptoms.**—The development of the characteristic symptoms is generally gradual, though it may rarely be rapid. In the so-called abortive form the symptoms arise somewhat rapidly, but early subside.

In *acute* Basedow's disease the symptoms consist of an excessively rapid action of the heart, incessant vomiting, purging, and marked exophthalmos, with or without pronounced cerebral symptoms. J. H. Lloyd's case proved fatal after an illness of three days.

In the *chronic* form heart-hurry is almost constantly a conspicuous early symptom, and not seldom have I found that it precedes for a long period of time the appearance of the remaining characteristic features. The pulse remains at or over 100 beats per minute, and upon unusual exertion or excitement the heart's action becomes violent and irregular, the pulse even reaching 160 or over. Palpitation, often with breathlessness, is a distressing symptom.

**Cardiac Physical Signs.**—*Inspection* reveals a forcible impulse that is not displaced, though late in the affection it may be much extended in superficial area. The carotids and the abdominal aorta beat more or less violently, and the capillaries and veins of the hands may also pulsate visibly. *Palpation* detects an increased force of the cardiac impulse. The area of *percussion dulness* may be somewhat increased, as hypertrophy and secondary dilatation supervene. On *auscultation*, blowing murmurs over the heart and the great vessels, as well as an increased accentuation of the valvular sounds, may be audible.

Protrusion of the eyeballs (exophthalmos) is usually present, and with rare exceptions follows the tachycardia. The degree of exophthalmos varies greatly from time to time in the same case—a fact that points to an increased amount of blood or lymph in the orbit as its cause. In advanced cases permanent prominence of the balls may be attributable to augmentation of the orbital adipose tissue. On closing the eyes a rim of

<sup>1</sup> *Mercredi méd.*, No. 34, 1895.

white is visible above and below the cornea, and Graefe's sign, immobility of the upper lid when the eye is turned downward, are two symptoms of great diagnostic importance. Möbius has called attention to the inability to converge the eyes upon near objects, and Stellwag to an apparent separation of the eyelids, due to spasm or retraction of the upper lid. The pupils and the vision are unaffected. Abnormalities are rarely presented by the optic nerves, and ulceration of the cornea may supervene. The retinal arteries pulsate.

The thyroidean enlargement either accompanies or follows the exophthalmos, and has for its cause the great dilatation of the vessels, and particularly of the arteries. The enlargement is usually moderate and may be general or partial, the size of the gland exhibiting sudden variations, since it is dependent upon the circulatory disturbance. Inspection may also show visible pulsation; palpation feels a thrill, and auscultation renders audible a double systolic murmur. The latter sign is probably present in most instances, though not constantly.

*Muscular tremors* form an early symptom; they are involuntary, and fine in character, numbering about eight to the second (Osler). The characteristic features of neurasthenia appear and gradually increase in intensity. Mental disturbances, particularly marked depression or great excitability, are common, and even mania (which may prove speedily fatal) or melancholia may be observed. Muscular weakness, either local or general, is pronounced; the patient becomes anemic and is at last extremely emaciated. The temperature may at intervals be moderately elevated, and this symptom may be associated with profuse sweatings. Among other cutaneous phenomena, though these are for the greater part occasional, are pigmentation (which, in the case of a physician whom I recently saw suffering from Basedow's disease, was as pronounced as in typical Addison's disease), scleroderma, urticaria, and circumscribed solid edema. In the advanced stage malleolar edema sets in and may become general. A marked diminution in the cutaneous resistance to the electric current has been noted by Charcot. Vomiting and purging may appear at different times and assume great gravity, and in some cases hemorrhages (epistaxis, hemoptysis, hematemesis) tend to supervene. Albuminuria and an increased amount of urine, with glycosuria, are among the commoner complications. Louise Bryson has maintained that diminution in the chest-expansion is a characteristic sign of exophthalmic goiter, and Patrick,<sup>1</sup> who examined 40 cases, found that there was an average diminution, but believed it to be proportionate to the amount of general muscular weakness. Rarely a myxedematous condition is associated; probably the disease is also remotely related to scleroderma.

**Diagnosis.**—The diagnosis of Graves's disease may be made when tachycardia or delirium cordis and fine, general muscular tremors are present. Exophthalmos and enlargement of the thyroid are often late-appearing symptoms, and are as often temporarily lacking even in fully-developed cases. Rarely, either or both of these signs may be permanently absent. On the other hand, in a few cases exophthalmos is the sole characteristic feature for a long time, though it is eventually followed by an unmistakable symptom-group. Parenchymatous goiter presents a non-pulsating tumor, and hence is easily distinguishable from the thyroid

<sup>1</sup> *Deutsche med. Woch.*, Dec. 20, 1894.



enlargement of Basedow's disease with its additional unequivocal symptoms.

**Course and Prognosis.**—The chronic form of the disease endures, as a rule, for a few years. A gradual subsidence of the cardinal symptoms for a long period has been noted, and in such cases complete recovery may be claimed. In fully-developed cases the prognosis formerly was almost hopeless, but since the introduction of the operative treatment many cases have been greatly benefited, and others, though constituting a smaller number, have been entirely cured.

**Treatment.**—This is (a) *Hygienic*, (b) *Medicinal*, and (c) *Operative*.

(a) **Hygienic.**—The environment, both physical and mental, should be made as favorable as possible. A change of climate, and especially moderate elevation, in cases not too far advanced, bring about beneficial results. Such elevation (3250 feet) produces a sedative effect upon the nervous state that reacts most favorably upon the circulatory organs, while the purity and tonic quality of the air have a general strengthening and restorative effect (Yeo). Among other promising measures may be mentioned the wet-pack, methodical hydrotherapy with massage, and a continuous galvanic current. The electric treatment should be given a thorough trial over three or four months (Osler). The local use of an ice-bag to the precordium has acted admirably in reducing the heart-hurry in a few cases of my own. I have also observed favorable results from carefully graduated physical exercise. Rest in bed for a few weeks at a time, at intervals, is often followed by improvement, though I have never seen complete cure follow this plan of treatment.

(b) **Medicinal Treatment.**—This is probably secondary to the hygienic and operative measures. In two cases of my own, however, recovery followed the persistent use, for about six months, of the following prescription:

R̄. Extr. digitalis,	gr. iv (0.259);
Extr. ergotæ (Squibb),	ʒss (2.0);
Strychninæ sulph.,	gr. ss (0.032);
Ferri arsenias,	gr. ij (0.129).

M. et ft. capsulæ No. xxiv.

Sig. One t. i. d. after meals.

In 2 other cases (one, a trained nurse) the use of sodium salicylate (gr. x—0.648—four times a day) was followed by almost total relief. L. Webster Fox also warmly advocates the latter remedy in this affection. Trachewsky, in Kocher's clinic, found that sodium glycerophosphate (gr. xx—1.296—three or four times a day), had the effect of diminishing the size of the enlarged thyroid glands, and Starr<sup>1</sup> has also found this remedy of great service in several cases. Other therapeutic agents that have been extensively employed, but with doubtful advantage, are aconite, veratrum viride, and belladonna. From all of the clinical testimony at hand I feel convinced that thyroid-feeding is contraindicated in the treatment of Basedow's disease, unless a myxedematous condition be associated, when it may prove efficient. From personal observation thyroid extract increases the circulatory disturbance and excites unpleasant headache.

(c) **Operative Treatment.**—Starr<sup>2</sup> has collected 190 cases in which

<sup>1</sup> *Medical News*, April 18, 1896.

<sup>2</sup> *Loc. cit.*



some form of operation was performed. Of these, 74 are reported as completely cured, many of them having been watched two to four years before the result was published; 45 of the cases were improved, and 23 died immediately after operation. The symptoms preceding the fatal result are sudden hyperpyrexia, with rapid pulse, nervous distress, sweating, cardiac failure, and collapse. The statistics of Kinnicutt and of Abram<sup>1</sup> (particularly the latter) show less encouraging results from operation, though they warrant the opinion that if cure is not obtained by medical measures, an operation should be undertaken. It is to be remembered that under the most favorable circumstances a complete cure will not be attained immediately, and frequently not for several years. I am convinced that removal of the entire gland is not to be advised, since myxedema will likely result. Whether partial removal—one-half to three-fourths of the gland—is to be effected, or mere ligation of the thyroid arteries, must be decided by the surgeon.

### MYXEDEMA.

(*Sporadic Cretinism.*)

**Definition.**—A general nutritional disorder, consequent upon atrophy and loss of function of the thyroid gland, and characterized by a myxedematous infiltration of the subcutaneous tissue and a cretinoid cachexia.

Three varieties occur, as follows: (1) True myxedema; (2) Cretinism (the absence of thyroid function—congenital, or lost during childhood); (3) Operative myxedema, due to total removal of the glands for surgical reasons or in experiments upon lower animals.

**Nature of Myxedema Proper of Adults.**—Charcot, who gave the name of *cachexie pachydermique* to this disease, believed it to be of tropho-neurotic origin. Atrophy of the thyroid is pretty constantly present, and the gland may either be converted into a small fibrous mass or be entirely absent, so that the causal relation between myxedema and functional and structural alterations of the thyroid seems to be conclusive. Moreover, the therapeutic test of improvement under the administration of thyroid extract sustains this view. It is probable that the active thyroid supplies some essential secretion which maintains normal metabolism, though this product has not been isolated. Its existence being inferred, however, it has been called *thyroidin*.<sup>2</sup> Others suggest that a substance called *thyro-proteid* is formed in excess in myxedema owing to a disturbance of glandular function, and this accumulating in the body produces the disease by a toxic action upon the metabolism.

**Etiology.**—The thyroid was destroyed by *actinomyces* in a case of myxedema reported recently. Myxedema may also be secondary to *exophthalmic goiter*, but it is then, as in the case of a simple acute goiter, only a transient condition. Women are much more frequently affected than men, and a neurotic condition may precede some cases. The disease may affect several members of a family, and hereditary transmission

<sup>1</sup> *American Year-Book of Medicine and Surgery*, 1897.

<sup>2</sup> The term "thyroidin" has also been given to a substance possessing specific therapeutic activities that has been obtained from the thyroid gland of the sheep by Baumann.

through the mother has been observed. Pregnancy may cause a disappearance of the myxedematous symptoms (Osler).

**Symptoms.**—The myxedematous condition is most plainly noted in the face, the skin being swollen, but inelastic, rough, dry, and firm. The lines of facial expression are obliterated, and the features are broad, coarse, immobile, and bulky. The physiognomy is stupid, dull, and phlegmatic, and simulates imbecility. The hair falls out, owing to deficient nutrition, and the general bulk of the body is markedly increased. Pressure does not produce pitting, as in true edema. According to Ord, the local tumefaction of the skin and subcutaneous tissue is most frequently prominent in the supraclavicular regions. The mucous membranes are also infiltrated, and the teeth may become loosened. The tongue, lips, and nose are thickened, and the voice is monotonous, slow, and has a "leathery tone," "with curious nasal explosions at short intervals during speaking." Bodily movements are slow, and the gait is heavy and uncertain on account of disturbed co-ordination. Mental perception and thought are also slow, and the memory is defective and slow to respond. Not infrequently there may be considerable irritability, or hebetude alternating with sudden excitability. The patient may become suspicious, and later is subject to delusions and hallucinations; or the apathy may pass into a melancholia, ending at last in dementia. Ord mentions "the aggravation of all symptoms during low climatic temperatures;" and "among the minor or accessory signs may be quoted abnormal subjective sensation, belonging particularly to taste and smell; occipital headache; marked alterations of temper; and a curious persistence of thought and action, overriding all attempts at interruption by friends or observers."

The temperature in myxedema is usually either normal or subnormal. Albumin and sugar are occasionally found in the urine, but the quantity of nitrogen excreted is small, owing to the diminished metabolism of proteids. Hemorrhages from the nose, gums, and bowels sometimes occur. Ascites also may be present in some cases, and may simulate ovarian tumor. The thyroid is not palpable, partly because of its atrophy, and partly because of the thickened myxedematous tissues of the neck.

The **diagnosis** is not difficult if one bears in mind the characteristic manifestations described above. Mxedema could hardly be mistaken for acute or chronic nephritis in the absence of pitting, etc., as some have supposed.

The **prognosis** is guardedly favorable in a majority of the cases since the introduction in the treatment of thyroid-feeding. The course of the disease is slow and progressive, however, often lasting from five to fifteen years, and death from intercurrent disease is not uncommon.

**Treatment.**—Until the advent of thyroid-feeding the treatment of myxedema was palliative, and usually unsuccessful.

A warm and equable climate is very desirable, owing to the subnormal temperature from which the patients frequently suffer. The various warm baths—as the Turkish, Russian, and electric—should be employed for the same reason. Pilocarpin has been recommended, and strychnin and arsenic have been administered for their tonic effect.



Since the brilliant results obtained by Murray, however, the internal use of the thyroid gland of sheep or calves has come into a well-deserved favor in the treatment of all cases of myxedema, whether of the so-called true form, of sporadic cretinism, or of the cachexia strumipriva. The gland may be given raw or cooked, in the form of the glycerin extract, or in the dried and powdered extract; the last named is sometimes put into tabloid form. If cooked, the gland should be only partially "done." The fresh thyroid is minced and often spread on bread, and from one quarter to half a gland may be taken daily.

The glycerin extract is readily made. "Several dozens of thyroids of young sheep or calves are carefully separated from the connective tissue, cut into small pieces about the size of a bean, and then put into a jar and covered with glycerin of the best quality, allowing 2 c.cm. of glycerin for each lobe of the thyroid used. The mixture is permitted to stand for twenty-four or thirty-six hours, and is then squeezed through a cloth, so as to get out as much liquid as possible. Of this, 2 c.cm., corresponding to about half a gland, may be given at a dose. If used for hypodermic injection, to a dram (4.0) of the glycerin extract is added half a dram (2.0) of a 1 per cent. solution of carbolic acid in distilled water, of which mixture from 10 to 15 minims (0.66–1.0) may be injected three or four times a week."<sup>1</sup>

It is safest—for reasons that will be pointed out below—to begin with quite small doses, and gradually increase, especially if there is much gastric irritation. Not more than 5 minims (0.333) of the glycerin extract should be given at the start. This dose may be increased gradually until 15 or 20 minims (1.0–1.33) are taken three times daily. From 3 to 5 grains (0.194–0.324) of the powdered gland or tabloid form will be a safe commencing dose in adult myxedema: a caution, however, is necessary regarding the various manufactured preparations of the thyroid gland, some of which are impure and even dangerous, owing to the careless handling or fraudulent substitution in order to meet the demand for thyroid extracts on trial in other affections (as obesity and psoriasis).

The toleration of thyroid-feeding does not depend upon the volume, but upon the functional activity, of the gland, and this fact, together with the evidences of toxic action reported in some instances of the administration of thyroids to a maximum degree, make it important to urge again—as intimated above—the necessity of small dosage at the beginning of treatment, the most careful and judicious increase in the quantity given, and the closest observation of symptoms indicative of hyperthyroidization. The additional fact of an occasional cumulative action should also be emphasized. Should vomiting, renal pain, tachycardia, suffusion of the face, syncope, vertigo, or marked headache supervene, the remedy should be stopped at once. Epileptiform convulsions have also occurred. The treatment may be resumed again cautiously, alternating with intervals of cessation. Good results are obtained usually within a month, though it is probable that even after all the symptoms have subsided the treatment may have to be continued off and on if the thyroid gland seems to be permanently atrophied.

<sup>1</sup> Osler in the *Amer. Text-book of Therapeutics*, pp. 926, 927.



**Cretinism, Sporadic and Endemic.**—Here there is a congenital atrophy or absence of the thyroid gland, or an enlargement by the growth of fibrous tissue at the expense of the glandular elements. Cretinism may also develop in early infancy. The patients are often the children of parents noted for violent emotion and having various neuroses and goiter, and syphilis has also been supposed to have a causative influence. Congenital myxedema is quite common only in regions where goiter is endemic, and hence it is rare in America. A marked sporadic case has, however, been in the Philadelphia Hospital for many years.

*Symptoms.*—Cretins are dwarfs with large heads and faces, thick lips, thick protruding tongues, broad bodies and members, and prominent abdomens. The subcutaneous tissues are myxedematous. Umbilical hernia has been noted. The mental condition is that of idiocy, and physical growth is retarded and slow. Speech is unintelligible or nearly so, and the voice harsh. Walking may never be accomplished, and is always slowly developed. There is anemia, the blood being of a fetal type. Rheumatic symptoms sometimes occur.

*Prognosis.*—The disease is progressive until about the fifteenth year in those cases developing during early childhood. Congenital cases usually die shortly after birth. At the twentieth or thirtieth year "the mental and physical characters are those of childhood."

*Treatment.*—Thyroid-feeding has been followed by beneficial results, the checked growth having recommenced and the cretinic aspect having been largely lost.

**Operative Myxedema, or Cachexia Strumipriva.**—Extirpation of the thyroid for surgical reasons has given rise to the gradual production of symptoms and conditions identical either with true myxedema or with the cretinoid state. Partial removal of the gland is not followed by cachexia strumipriva, nor is complete thyroidectomy when accessory glands are present elsewhere.

The administration of raw or broiled thyroids, or of their various extracts or preparations, must also be employed in this form of myxedema, and should be continued throughout the rest of the patient's life, perhaps with intervals of withdrawal of the feeding until the improvement gained begins to lapse.

## PART IV.

# DISEASES OF THE RESPIRATORY SYSTEM.

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## I. DISEASES OF THE NOSE.

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### ACUTE RHINITIS.

(*Acute Nasal Catarrh; Acute Coryza.*)

**Definition.**—An acute catarrh of the Schneiderian membrane, sometimes tending to involve the adjacent sinuses and passages. It is known to the laity as “cold in the head.”

**Etiology.**—Its most conspicuous cause is exposure to draughts of air and to the influence of the atmospheric vicissitudes that are especially prevalent during the winter and spring seasons. It often results from the inhalation of irritants (physical, chemical, or biological). It may also display epidemic behavior, and this fact seems to point strongly to its microbic origin. Hence local disturbances of the circulation due to exposure are to be regarded as the accidental means of preparing the soil for bacterial invasion. Acute rhinitis may be also secondary to, or propagated from, inflammations of the faucial mucosa by contiguity.

**Symptoms.**—Sensations of chilliness, succeeded by feverishness (the temperature reaching 100° to 101° F.; 37.7°–38.3° C.), frequent sneezing, headache, and a feeling of general ill-health are among the prominent features that attend the development of coryza. Pains in the extremities and back tend to appear only in severe cases. The pulse is frequent, the skin dry and unduly warm, thirst is increased, while the appetite is impaired, and constipation often attends. The nasal mucosa is swollen, and thus interferes both with the nasal respiration and the senses of smell and taste; its color is deepened, its surface covered at first with opaque mucus, and later with a muco-purulent secretion. Among early symptoms is the discharge of a watery, irritating secretion from the nares and a maceration of the epidermis, with resulting abrasions. On account of the swelling of the mucosa of the lacrymal ducts the tears flow down over the cheeks. Adjacent mucous surfaces then become involved, giving rise to conjunctivitis, catarrhal pharyngitis, laryngitis, and finally, in the severer types, bronchitis. Naso-labial herpes is not uncommon. As the affection progresses the secretion becomes more abundant and turbid and more or less pyoid. The symptoms due to extension of the catarrhal inflammation vary with the organs or structures involved. The disease runs its course within five or six days, but

the nasal discharge, which gradually diminishes, usually persists for a few days longer.

**Diagnosis.**—In the presence of the above-mentioned symptoms the disease is readily recognized. In well-marked cases, however, the possibility that an infectious disease may be developing, the beginning of which is characterized by nasal catarrh (measles, influenza, etc.), is to be recollected.

**Prognosis.**—Except in neglected cases, which result in bronchitis, and occur at one or other extreme of life, the disease is free from danger. The nursing infant may have to be fed with a spoon temporarily.

**Treatment.**—At the outset a purge, consisting of calomel (gr. ij—0.129), or a pill of blue mass (gr. v—0.324) at night, followed by a Seidlitz powder in the morning, is advisable. To children a dose of castor oil may be given. The early administration of a diaphoretic, such as Dover's powder (gr. v—x—0.324–0.648) at night may arrest the complaint, and quinin in a large dose (gr. xij—xv—0.777–0.972) at night may cut short the course of the disease. When the above-mentioned abortive measures fail, the following tablet produces good results:

R. Quinin. sulphat.,	gr. ijss (0.162);
Extr. belladonnæ fl.,	℥jss (0.099);
Sodii salicylatis,	gr. xxx (1.944);
Camphoræ,	gr. ijss (0.162).
M. et ft. tablet No. x.	

Sig. One tablet every hour or two.

For the fever aconite may be employed, and, if the throat be involved, bryonia may be given in conjunction.

**Local Treatment.**—This aims at soothing as well as at reducing the swelling of the Schneiderian membrane. The compound tincture of benzoin forms a soothing inhalation (ʒij to a pint—8.0 per half liter—of water) when raised nearly to the boiling-point; the vapor is inhaled for ten or fifteen minutes at a time. With a view to reducing the swelling a solution of cocain (strength 2 to 4 per cent.) may be temporarily used; Mackenzie recommends this admirable combination:

Menthol,	gr. v (0.324);
Pinol	℥v (0.324);
Benzoinol,	fʒj (32.0).

In severe cases the patient should be kept in-doors and in an atmosphere of even temperature.

## CHRONIC RHINITIS.

(*Chronic Nasal Catarrh.*)

Two forms are recognized, the hypertrophic and atrophic, and these, though, as a rule, occurring separately, may be found in combination.

**Pathology.**—The morbid changes in hypertrophic rhinitis consist in an enlargement of the lower turbinated processes, together with redness and swelling of the nasal mucosa that may be general or limited



either to the anterior or posterior nares. As the disease progresses the thickening of the membrane increases, until it finally encroaches upon the nasal chambers at every point. In addition to the nasal obstruction there is a hypersecretion of mucus. Opposite changes occur in atrophic rhinitis, such as thinning or atrophy of all the structures, with enlargement of the nasal cavities. The nasal mucosa is coated with thick, yellowish-green, decomposing crusts, which emit a characteristically fetid odor, and the frontal, ethmoid, or other accessory sinuses may, by an extension of the inflammation from the nasal chambers, be invaded by mucopurulent inflammation. The atrophic process does not affect the glandular structures of the upper third of the nose, and this fact explains the most unpleasant feature of the affection—namely, the horrible secretion.

**Etiology.**—Frequently occurring attacks of acute rhinitis may produce the chronic form, and syphilis and, less commonly, tuberculosis are also among its causes. Abel<sup>1</sup> regards atrophic rhinitis as infectious, claiming that the cause is the *bacillus mucosis ozenæ*, which resembles closely the pneumobacillus, but is distinguishable from it.

**Symptoms.**—(a) In the *hypertrophic* form nasal respiration is impeded, owing to the hypertrophy of the turbinated bodies. The sense of smell is impaired, and there is a discharge of secretion from the nares, particularly the posterior, inducing “hawking.” The diagnosis is set at rest by a rhinoscopic inspection of the parts. While this is a common affection everywhere, it is wellnigh universal in this country.

(b) In chronic *atrophic* catarrh there is some degree of nasal obstruction, occasioned by the presence of the thick crust, but the most conspicuous symptom is the disgusting odor, which makes the patient repellent in society. The sense of smell is lacking. After cleansing the membrane the rhinoscope will show the nasal chambers to be unduly capacious.

**Treatment.**—(1) **Chronic Hypertrophic Rhinitis.**—The treatment is divisible into *general* and *local*. The physician should procure an environment for his charge most favorable for promoting the general nutrition, which is often below the health-standard. The selection of a suitable climate, then, forms an important part of the management, and a residence in a locality that possesses a mild, equable, comparatively dry and pure atmosphere is to be advised and encouraged. Various tonics may then be demanded by the general condition of the patient, and strychnin and electricity are useful in restoring the loss of power in the contractile elements of the intercellular walls.

Local measures are employed to facilitate thorough cleanliness and



FIG. 41.—Apparatus for cleansing the nasal passages in chronic rhinitis.

<sup>1</sup> *Zeit. f. Hyg. u. Infektionskrankh.*, Bd. xxi. H. 1.

disinfection of the affected parts, though in incipient and mild cases energetic treatment is scarcely needful. The best method of cleansing the nasal passages is by means of the coarse spray (Fig. 41). The apparatus of Lefferts is also to be employed when the secretion is inspissated or tightly adherent. An excellent combination for use in this manner is the following:

R<sub>x</sub>. Sodii biborat.,  
 Sodii bicarb.,                      *aa*. ʒj            (4.0);  
 Acid. carbolic,                      gr. viij    (0.518);  
 Listerin.,                              ʒj            (32.0);  
 Aquæ destillat.,    q. s. ad ʒiv    (128.0).—M.

Sig. Use as a spray three times daily.

It is often desirable to use warm or even hot liquids, in which case the application is made by the use of the anterior and posterior nasal syringe. Powders are harmful, and, as the nasal douche is dangerous in unskilled hands, these should both be abandoned.

In hypertrophic rhinitis the obstruction to nasal breathing is to be removed, and to accomplish this caustics (chromic, glacial acetic, and nitric acids) are used, of which the most efficacious is chromic acid. This should be applied by means of a pointed glass rod, the application being followed by a sloughing away of the diseased tissues. Among other modes of removing the nasal obstruction that may be mentioned are the galvano-cautery, the thermo-cautery, and the cold-wire snare; these modes, however, are practised chiefly by the specialist.

(2) In **atrophic rhinitis** a cure is to be despaired of, but the patient can be rendered free from the offensive discharge, and hence to a great degree comfortable. As this is often but an advanced stage of hypertrophic nasal catarrh, the general treatment is similarly directed: it is therefore well to overcome, as far as possible, by a mental stimulus, the depressed mental state due to the fetor. If the diathesis be tuberculous, cod-liver oil, iron, arsenic, and strychnin, together with a generous diet, are to be advised. If syphilis is associated, appropriate measures must be instituted. Moreover, since a subject of atrophic rhinitis is a fertile source of atmospheric contamination, his living and sleeping apartments must be highly ventilated.

*Local Measures.*—An antiseptic spray of Seiler's or Dobell's solution, and oiling the nasal cavities, are measures to be first tried. If they prove non-efficacious, the crusts may then be removed with a cotton applicator coated with a solution of hydrogen peroxid. We may then use a spray of liquid albolene and menthol; this serves not only to lubricate, but to supply moisture, both of which are important therapeutic indications. Small ulcerations occur in this affection and induce oft-repeated epistaxis; consequently, an attempt should be made to heal the latter and to obtain an even, moist surface. To accomplish this the method of Clarence C. Rice may be followed—*i. e.* to rub the ulcerations thoroughly by means of a cotton-carrier with a small hard pledget of cotton moistened with listerin or borolyptol for a few seconds at a time. These antiseptic frictions are made at intervals of two or three days for two or three weeks.

## AUTUMNAL CATARRH.

(*Hay Asthma ; Hay Fever.*)

By this term is meant a form of asthma that seems to be dependent upon an idiosyncrasy. It occurs exclusively during the warm season, and is caused by the odorous principles given off from certain plants (the pollen of the *Anthoxanthum odoratum*, of the rose, etc.), by inorganic dusts of various sorts, and, occasionally, by psychical influences. In some instances it appears to arise without obvious exposure to a special irritant.

**Predisposing Factors.**—The male *sex* suffers more frequently than the female. *Age* has a slight though decisive influence, more than 33 per cent. of the cases occurring before the twentieth year. The inhabitants of cities are more liable than those in rural districts, though the air of agricultural regions intensifies the condition. Perfect immunity is enjoyed by the dwellers in certain climates—chiefly mountainous and marine.

**Symptoms.**—The symptoms are (*a*) local and (*b*) general.

(*a*) **Local.**—Hay fever has an abrupt onset, and the attacks return annually at or about the same time. The invasion is marked by pronounced coryzal symptoms, with much sneezing, stoppage of the nasal passages, copious rhinorrhea, the discharge being thin and watery as a rule, and rarely mucopurulent. Suffusion of the eyes, with itching of the lids and free lacrymation are constant features; the decided itching sensation of the palate and pharynx is also at times a very distressing symptom. The sense of smell may be lost, and taste and hearing are often impaired.

The course as regards the local symptoms is marked by alternate amelioration and aggravation of the symptoms, the exacerbations being due to exposure to the open air, especially in changeable weather. Later the catarrhal process invades the bronchi, and cough and asthmatic seizures appear, these often becoming very distressing.

(*b*) **General disturbances** are varied, and comprise subjective sensations, such as anorexia, insomnia, lassitude, and chilliness alternating with slight feverishness.

The course is usually run in from four to six weeks, and cases that occur in the early autumn are usually terminated speedily by the occurrence of a decided frost.

**Diagnosis.**—The recognition of hay asthma is unattended with difficulty, provided that such facts as the time of their occurrence and their annual periodicity are carefully noted.

**Prognosis.**—This is favorable both as to life and length of days, though a permanent cure is among the rarest events in medicine unless permanent removal from the influence of the specific causes can be effected.

**Treatment.**—Whenever possible the patient should travel till he finds a locality in which he ceases to suffer, and subsequently he should there spend the period of annual attack, and by these means escape the exciting causes. The Adirondacks and White Mountains usually bestow immunity. If the patient cannot make the necessary change, the gen-



eral nutrition is to be improved by the use of such measures as phosphorus, strychnin, quinin, and arsenic. Much is to be gained, moreover, by hygienic means, especially avoidance of physical and mental overwork and the adoption of a proper mode of life.

The local symptoms demand the topical application of various agents to the nasal chambers. A solution of cocain hydrochlorate (1 per cent.), applied directly to the nasal passages by means of a probe, around the end of which is loosely wrapped a little absorbent cotton, affords temporary relief, and the period of palliation may be very much prolonged by using a 4 per cent. solution of antipyrin immediately after the cocain solution (Gleason). These applications should be made two or three times daily, according to the severity of the individual case. The local symptoms are also greatly benefited by the internal use of atropin, which allays the irritability of the mucous membrane involved and diminishes the rhinorrhea, thus indirectly mitigating the constitutional disturbances and sometimes directly relieving the asthmatic paroxysms. When given internally the dose should not exceed gr.  $\frac{1}{300}$  (0.0002), to be repeated every hour till dryness of the throat appears.

My own best results have been derived from the hypodermic use of this drug (gr.  $\frac{1}{200}$ —0.0003) at intervals of three to four hours till the desired effect is produced. Thorough destruction of the vessels and sinuses is also advised (Osler).

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## EPISTAXIS.

(Nose-bleed.)

**Etiology.**—The causes of nose-bleed are various, and a convenient grouping is the following: (a) Affections of the nasal mucosa (*e. g.* ulcer, polypi, intense hyperemia). (b) Injuries, either external, as from a blow, or internal, as from plugging with a foreign body, nose-picking, etc. In this category may also be included epistaxis due to fracture at the base of the skull. (c) Acute infectious fevers, particularly typhoid (at the onset) and influenza. (d) Chronic affections, such as pernicious anemia, leukemia, arteriosclerosis (with which cardiac hypertrophy is associated); also the hemorrhagic diathesis or hemophilia. (e) Vicarious menstruation. (f) Rarefaction of the air. (g) Plethora; here may be mentioned cerebro-congestion with intense headache. (h) Severe over-exertion.

**Symptoms.**—Except when due to traumatism the blood usually drops slowly from one and occasionally from both nostrils. Rarely, the blood may flow as a continuous stream or the nares may present a projecting coagulum. The blood may also gravitate into the pharynx and be coughed up, or it may be swallowed and vomited. A rhinoscopic examination often reveals the source in cases in which a previous diagnosis of hemoptysis or hematemesis has been made.

The immediate results of nose-bleed are weakness and a moderate anemia, but these are not prolonged unless the epistaxis be oft repeated. Cases arising from fracture at the base of the skull will generally prove fatal.

**Treatment.**—A careful search for a local cause is especially demanded in cases in which there are frequently recurring attacks. In most cases a spontaneous arrest occurs, but if not, a resort to simple household measures, such as the application of ice to the nose or to the back of the neck, holding the hands up, or the injection of very cold or very hot water into the nares, are to be encouraged. Various astringents (tannic acid, acetate of lead, alum, zinc) may be employed, and a saturated solution of antipyrin is also highly praised. When an ulcerated bleeding point can be reached, there may be applied to it a solution of chromic acid or it may be cauterized by solid silver nitrate. Prolonged pressure applied upon the facial artery as it passes over the inferior maxilla may be efficacious. The late D. Hayes Agnew successfully employed a bougie made of a long strip of the rind of bacon, "passing it through the nostril and allowing it to stay there some time." I have little confidence in internal astringent remedies, such as ergot, acetate of lead, or gallic acid, as a means of arresting nose-bleed. The oil of *origanum*, administered in large doses, has seemed to do good in a few of my own cases, but when the epistaxis tends to prove obstinate, the posterior nares should be plugged.

## II. DISEASES OF THE LARYNX.

### ACUTE CATARRHAL LARYNGITIS.

(*Acute Endolaryngitis.*)

**Definition.**—An acute catarrhal inflammation of the larynx, characterized by cough, hoarseness, and painful deglutition.

**Pathology.**—The anatomic changes present during life are all lacking *post mortem*. The laryngoscopic appearances will be given among the Clinical Symptoms.

**Etiology.**—Acute laryngitis may be a primary affection—and particularly *laryngitis sicca* (Molinie)—but oftener it is associated with and secondary to catarrh of the nose and nasopharynx. Wright attributes *laryngitis sicca* to the coccus of Löwenburg.

Catarrhal laryngitis has for its chief direct causes traumatism, exposure to cold and dampness, the inhalation of irritating vapors or gases, rheumatism (rarely), and the corrosive effect of certain poisons and hot fluids. A certain degree of predisposition is engendered by immoderate smoking, particularly by the cigaret-habit, and by the use of concentrated alcoholic drinks. These agencies induce hyperemia of the laryngeal mucosa, which is easily converted into active inflammation. Acute laryngitis is often associated with acute infectious diseases.

**Symptoms.**—There are two conspicuous symptoms—*alteration in the voice* (hoarseness) and *cough*. At first there is merely a huskiness of the voice, but later there may be pronounced hoarseness or even complete aphonia. The cough is dry and characteristically painful until secretion is free. In the early stages the patient complains of sensa-

tions of tickling or the presence of some small object in the larynx, causing a frequent desire to clear the throat. In severe instances deglutition is painful. Edema of the larynx may tend to supervene and cause intense dyspnea, with a feeling of distressing oppression. There is, as a rule, a slightly elevated temperature.



FIG. 42.—Method of making a laryngoscopic examination.

The patient is placed in front of the operator, on an arm-chair, with the back of the chair high enough to afford his head a comfortable rest, and with the source of the light over the right shoulder. The operator then adjusts the head-mirror (the fixed apparatus), warms the throat-mirror over a light sufficiently to prevent the moisture of the breath from being deposited upon it, and touches the hand with the mirror before passing it into the mouth, so as not to use it too hot. The patient's tongue is then protruded, and by means of a napkin is seized between the thumb and the fore-finger and drawn well forward to lay the fauces open to observation. The throat-mirror is then held in the right hand in the same way as one holds a pen. "Finally, it is introduced into the mouth, its handle being inclined downward and outward, its base being parallel with the dorsum of the tongue; it is then passed backward without altering this relation until the edge of the mirror nearly touches the soft palate, the shaft of the mirror in this movement striking the angle of the mouth as a resting-place and fulcrum. The subsequent movement consists in turning the mirror by twisting its shaft between the fingers until it is inclined at an angle of 45 degrees to the line of vision; then it is carried backward and downward until the uvula rests upon its posterior surface, when it is lifted boldly upward and backward until its lower edge comes entirely into view again and rests firmly against the posterior wall of the pharynx. The patient should then be directed to sound in a somewhat high key 'a,' which lifts the larynx and at the same time the epiglottis, and exposes and brings into view the laryngeal cavity" (Bosworth).

It is important that the mirror itself should be kept in the median line, with its plane always at right angles with the field of vision, as shown in the illustration. In making a laryngoscopic examination we note any abnormalities of color-appearance (the natural being a rose-pinkish tint), of the outline of the different parts, and the deviations from the symmetrical movements of cords, if any, etc.

The laryngeal mirror brings to view a characteristic picture—a swollen, tumefied, and reddened mucosa. These changes affect the vocal cords (whose pearly-white appearance is now lacking) and the ary-epiglottidean folds. It is usual to note also redness and swelling of the epiglottis above and of the trachea below. After secretion has occurred a mucoid covering in streaks or patches is noticeable.



**Diagnosis.**—This is easy in the presence of marked hoarseness, dry cough, and the image afforded by the laryngeal mirror (Fig. 42). In very early life the larynx cannot be successfully examined; still, *laryngismus stridulus* (owing to the absence of fever, coryza, etc.) could hardly be mistaken, as has been supposed, for acute catarrhal laryngitis. The same is true of *membranous laryngitis*, if we bear in mind the characteristic local features and the more intense constitutional disturbances of the affection.

**Treatment.**—The physician must enjoin against the use of the voice. The very young and the aged should, in severe or even moderate cases, be kept in bed, and should occupy a single apartment in which the atmosphere is uniformly moist and warm, the temperature ranging from 75° to 80° F. (23.8°–26.6° C.). Inhalations of moist air or steam are of great service, and I have long been in the habit of recommending the following simple apparatus and method of carrying out this mode of treatment: An ordinary tin cup, small pitcher, or other vessel is filled with boiling water to which 1 or 2 drams (4.0–8.0) of the compound tincture of benzoin have been added; the steam is then collected by inverting over the vessel an ordinary funnel. The patient is allowed to inhale the steam by placing the mouth over the narrow neck of the funnel above, or a piece of rubber tubing may be attached to the end of the funnel that is uppermost.

Steam atomizers admirably meet the necessities of the case; and in the case of children the vapor of benzoin, eucalyptol, and other equally sedative and stimulating substances may be diffused in the air of the sick-room. Concentrated solutions or insufflations of powders are not without harmful influence, and neither the cotton-carrier nor the mop should be allowed to enter the larynx in this affection. The external application of the ice-bag or cold compress tends to mitigate the inflammatory process and to obviate spasm.

The *general* treatment differs with the special stages of the complaint. If the case is seen early, a full dose of quinin (gr. xij–xvj—0.777–1.036) may serve to successfully abort the attack, and, in conjunction Dover's powder (gr. v–x—0.324–0.648) may be prescribed. Codein sulphate may be given at prolonged intervals during the attack, and frequently at night, to allay cough; this remedy may be combined with ipecac, aconite, and liquor ammonii acetatis to facilitate secretion and render the cough humid. If we except the abortive measures, the constitutional is wholly inferior to the topical treatment of this variety, though the existence of any particular diathesis may require special internal remedies.

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## CHRONIC LARYNGITIS.

(*Chronic Endolaryngitis.*)

**Pathology.**—The laryngeal mucosa is thickened and somewhat reddened, and erosions amounting to superficial ulcerations are sometimes seen, though they are infrequent. A prominence of the mucous glands, especially of the ventricles and epiglottis, is noticeable. Fine villous

projections from, and nodular swellings in, the vocal cords are among the rarer morbid changes.

**Etiology.**—Of repeated acute attacks frequently cause chronic laryngitis, and the long-continued use of the voice (as in public speaking or singing), the inhalation of an atmosphere laden with mildly irritating impurities (tobacco smoke, etc.), and an immoderate indulgence in alcoholic stimulants, respectively or unitedly, predispose to, if they do not excite, the disorder.

**Symptoms.**—As in the acute form, hoarseness and cough are the two especially prominent symptoms. The former may be so slight as to present merely a rough tone, or it may involve an almost total loss of voice. The cough shows similar variations in severity, sometimes consisting of a short hack, and again occurring in spasmodic and ringing paroxysms, due to a sense of tickling in the larynx. There may be a small amount of mucous or muco-purulent expectoration, but for prolonged periods the cough may be dry and ineffectual. Local pain and discomfort sometimes supervene, and are excited generally by attempts at speaking or singing—events that aggravate all the other symptoms. To complete the diagnosis, the laryngeal mirror is required to show a swollen and slightly red membrane, with a distention of the mucous glands in the immediate vicinity of the epiglottis and ventricles, and occasionally superficial erosions.

**Prognosis.**—This is unpromising as to complete recovery, although it presents no grave dangers. It is incurable in those instances in which the causal influences cannot be removed, and in all cases in which the patient fails to lend hearty co-operation.

**Treatment.**—This is (a) *hygienic* and (b) *medicinal*. (a) The sanitary measures embrace preventives that are directed to the removal of all the etiologic factors, whether merely predisposing or exciting. The voice demands rest and the prohibition of smoking and the use of alcoholics in excess, and the patient must also avoid the close, contaminated air of the crowded hall, theatre, and like places. In addition, a tonic regimen, with a view to energizing the nutritive processes, is to be encouraged. In many instances the environment is best arranged with reference to the commonly associated conditions—especially the morbid processes in the nasal and naso-pharyngeal cavities. “A sea-voyage or residence at the sea-shore is, in the large majority of instances, productive of good, and the effects of surf-bathing are often magic” (Mackenzie). My own practice has been to send subjects of chronic laryngitis to pine-forest resorts at low elevations that afford a pure, equable, and somewhat stimulating atmosphere, and I have found that in many cases the selection of a proper climate constitutes the most important part of the treatment. (b) The *medicinal* treatment is both local and general. The latter should include creasote, cod-liver oil, and other tonics. Expectorants are of little if any value. The *local* measures, however, are important. Moderate exposure of the neck and daily ablution with cold water are to be advised, and attention to the nose and naso-pharyngeal cavity is of prime importance.<sup>1</sup>

A long list of applications to the larynx from within, including local astringents, disinfectants, and alcoholics, might be enumerated. Of

<sup>1</sup> J. C. Wilson's *American Text-book of Applied Therapeutics*, p. 791.



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 astringent solutions, however, the best are tannic acid (1–2 per cent.) or alum (.5–1 per cent.) and zinc sulphate (3–5 per cent.). These may be sprayed into the larynx by means of a compressed-air machine with spraying-tubes, although all of the different kinds of inhaling apparatus more commonly used will answer the purpose. If the ordinary hand-atomizer be used, the patient should be taught to draw the vapor into the larynx by gentle and frequent acts of respiration. Disinfectants, such as creasote, potassium chlorate (the latter if ulcerations be present) in solutions of suitable strength, may be used in like manner. I can confidently advise as useful alteratives both iodine and silver nitrate, commencing with a weak solution of the latter (gr. v–3j—0.324–4.0), and the strength being gradually increased until the maximum strength that can be endured without distress is reached (gr. xx–3ij—1.296–8.0). These topical applications should be made directly with a cotton-carrier or brush at intervals of three or four days, preceded by the use of a cleansing spray. Many astringent and sedative lozenges are to be found in the market, but they are only slightly palliative in their effects, and their prolonged use tends to excite gastric disturbance. I am unalterably opposed to the insufflation of powders, believing that they are capable of augmenting the laryngeal irritation and of adding fresh irritation in adjacent parts, particularly in the tracheo-bronchial tract.

## SPASMODIC LARYNGITIS.

(*Laryngismus Stridulus*; *False Croup*.)

**Definition.**—An affection peculiar to children, chiefly of nervous origin, though also, according to Strümpell and others, often associated with acute catarrhal laryngitis.

**Etiology.**—The affection is almost solely limited to children between six months and five or more years of age. It is sometimes excited by strong passion or emotion, and it may be associated with tetany. Rachitic subjects are peculiarly liable. The causes of spasmodic croup are in great part those of acute laryngitis.

The mode of action of the direct causes is unknown, but the spasm of the adductors that causes the urgent dyspnea is probably reflex and due to peripheral irritation.

**Symptoms.**—Two clinical varieties are to be distinguished: (1) that in which the larynx is free from catarrhal inflammation, or the *purely nervous type*. This is especially characterized by sudden brief attacks of dyspnea, either by day or night (often on awakening), that terminate in a high-pitched crowing inspiration (“child-crowing”). The face during the spasm is cyanotic. General convulsions have been noted, but there is neither cough, fever, nor hoarseness. The attacks may be frequently repeated within a single day.

(2) Spasm of the larynx, *associated with mild catarrhal laryngitis*. The attacks generally begin suddenly, about midnight or toward morning on awakening from a sound sleep. Positive evidence of the affection is afforded by the croupy, ringing cough, combined with the hard, stridulous breathing. An approaching spasm may be announced by a harsh



cough and slightly stridulous breathing in the sleeping child. During the attack the countenance may be cyanotic and the breathing most distressing, but these and the above-mentioned severer symptoms generally cease abruptly in an hour or two, and the child resumes its slumber. In my experience the attacks have been repeated for two or three nights in succession, and rarely oftener except in the severest cases. Not infrequently the child manifests the symptoms of mild catarrhal laryngitis between the attacks. A brassy, croupy cough may also attend.

**Diagnosis.**—*Membranous laryngitis* may be mistaken for spasmodic croup. The development of the dyspnea, however, is more gradual, is without intermission, and without relation to the period of the day. Albuminuria and a false membrane in the throat or nares are usually present in *laryngeal diphtheria*.

**Prognosis.**—Although the appearance of a paroxysm is alarming, the disease is practically free from danger.

**Treatment.**—1. The treatment of laryngismus stridulus is quite similar to that of infantile convulsions. A warm bath at a temperature



FIG. 43.—Croup-kettle in use.

Four upright rods (5.7 inches in length) are fastened to the legs of the bedstead by a wire or string. Two side-rods are tied on the uprights, and two end-rods (length dependent on width of bed) rest upon the side-rods. These rods form a complete framework for the sheets to hang upon. Four sheets are required (11-4 size)—three to cover the ends and sides, and one to be placed on top. One side should be completely closed, while the opposite is to be left open for ventilation or to be adjusted according to circumstances.

of 98° to 105° F. (36.4°–40.5° C.) is the best means of breaking up the spasm. While in the bath cold sponging of the back and chest is serviceable. The finger may be passed into the fauces, and should the epiglottis “become wedged in the chink of the glottis, it must be released by the finger.” After the attack active treatment should be directed at the discoverable causes, and I have been in the habit of giving

small doses of the bromids thrice daily, together with warm cod-liver oil inunctions, with striking effect.

2. In spasmodic croup an emetic is to be given at once, the best being a mixture of alum and syrup of ipecac, of which the dose is 3j (4.0), to be followed by irritation of the fauces with the finger in order to facilitate emesis. In severe paroxysms a hot bath may be given to aid the emetic. In case the dyspnea is not checked by the above measures, chloral hydrate may be exhibited by enema (gr. ij-v; 0.129-0.324) or a whiff of chloroform may be given. The local application of cold (ice-collar, ice-water cloths) is useful, and sinapisms placed around the throat and over the chest also tend to arrest the spasm. I am convinced that the use of steam-inhalations from the so-called croup-kettle (Fig. 43) is of signal service, and should be more widely employed, particularly when it is inconvenient to use the hot-bath.

Between the paroxysms the patient should receive a mild laxative, such as calomel or castor oil, and, in addition, the treatment appropriate in acute catarrhal laryngitis. To prevent recurrences an environment calculated to increase the nervous tone of the child is to be procured, and it is especially advisable to accustom him to the outer air, though protected by suitable dress and without undue exposure to draughts.

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## TUMORS OF THE LARYNX.

THESE may be either benign (fibroma, myxoma, lipoma, chondroma, adenoma, angioma, cyst) or malignant (sarcoma, carcinoma). Of these, fibroma occurs most frequently, and with an especial frequency in infancy. Navratil<sup>1</sup> records 42 cases of multiple laryngeal papilloma in children whose larynges were extensively filled. These growths may also occur in chronic laryngitis, and, like other tumors of the larynx, they commonly spring from the vocal cords. Their shape, size, and tendency to pedunculation do not differ from their characteristics when noted elsewhere in the body.

**Symptoms.**—Small tumors may occupy the larynx without producing symptoms. The first feature then noted is hoarseness, which gradually grows worse and may end in complete aphonia. If situated in the upper larynx, cough is common, and when the tumor causes obstruction of the larynx dyspnea supervenes and tends to increase in severity. A mobile growth may cause sudden occlusion of the glottis, exciting orthopnea and threatening asphyxiation. To confirm the diagnosis a laryngoscopic examination is required.

The **prognosis** is favorable in the benign, but unfavorable in the malignant forms.

**Treatment.**—This is altogether surgical, though Delavan states that 3 cases of papilloma have been cured by frequently repeated sprays of absolute alcohol. Curetting is often followed by a recurrence, while laryngo-fissure and thorough removal of the growths restore speech and prevent recurrence.

<sup>1</sup> *Berl. klin., Woch.*, Mar. 9, 1896.

## EDEMA OF THE LARYNX.

**Definition.**—An infiltration of the mucous membrane of the larynx with serum, affecting mainly the region of the epiglottis and of the ary-epiglottic folds.

**Etiology.**—Two chief classes of causes are operative: (1) Those that excite inflammation. The condition may complicate acute laryngitis, though oftener it appears in chronic affections of the larynx, and particularly if ulceration be associated (*e. g.* tuberculosis, syphilis); it may also appear in connection with certain infectious diseases, such as erysipelas or diphtheria. The inflammation inducing the edema may extend from adjacent parts, as the neck, pharynx, and other organs. (2) Factors that tend to excite dropsical effusion. These may be general, as Bright's disease, which may cause a quickly developing edema of the lungs, heart-affections, etc.; or they may be local. Among the latter are enlargements of the cervical and mediastinal lymphatics, aneurysm of the arch of the aorta, thyroid tumors, etc.—*i. e.* conditions that exercise pressure upon the jugular veins.

**Symptoms.**—In acute cases the initial disturbance is both sudden and severe. There is dyspnea that tends to increase rapidly, accompanied by a husky, suppressed voice, with augmenting obstruction. The respiration may become stridulous, but there is no cough. The laryngoscope reveals marked swelling of the epiglottis and of the ary-epiglottic folds, and rarely the swelling occurs in or even wholly below the vocal cords. The inserted finger may detect the swollen epiglottis, which may also be seen if the tongue-depressor be used.

**Diagnosis.**—This can be made with ease from the rapidly developing dyspnea soon reaching the climax, the absence of cough and hoarseness, and by the use of the laryngoscope. In cases in which the epiglottis can be felt or seen a laryngoscopic examination is superfluous.

The **prognosis** is decidedly unfavorable except in the event of early operative interference.

**Treatment.**—If of inflammatory origin, the ice-bag should be applied to the larynx, and ice should be allowed to constantly dissolve in the patient's mouth. Local depletion, preferably by leeching the front of the neck, is also to be tried, and Levy and Laurens<sup>1</sup> record a case in which a cure followed this measure. If intense dyspnea tends to persist, scarification of the edematous parts with a curved bistoury, the point of which is covered with adhesive plaster, must be promptly instituted, and, if asphyxia threatens, tracheotomy must immediately be performed. Dropsical edema demands scarification, and, if relief does not follow, tracheotomy.

## III. DISEASES OF THE BRONCHI.

## CATARRHAL BRONCHITIS.

(*Tracheo-bronchitis.*)

**Definition.**—A catarrhal inflammation of a part or the whole of the mucous membrane of the bronchial tubes. The mucosa of the

<sup>1</sup> *Arch. gén. de Méd.*, Dec., 1895.



trachea is also involved to a greater or lesser extent, and hence the term tracheo-bronchitis is quite appropriate, being descriptive of the seat and character of the disease. Involvement of the bronchioles may also take place, but this does not occur without an involvement of the corresponding alveolar structure, the condition being then, with propriety, termed "broncho-pneumonia." Hence the term "capillary bronchitis," which is still often employed to describe the latter condition, is not pertinent. A certain class of cases is met with, however, in which the catarrhal inflammation, as the result of downward extension, implicates the smaller bronchial tubes without involving the bronchioles; to such the term "capillary bronchitis" might be appropriately given.

The disease may be acute or chronic, both of these forms occurring either as a primary or secondary affection.

#### ACUTE BRONCHITIS.

**Pathology.**—The portions of the mucous membrane of the trachea and bronchi that are implicated become reddened and swollen; they are covered with mucus and mingled with epithelial cells, and later mucopus. Some of the smaller bronchial tubes are dilated. The mucous glands are swollen.

The histological changes may be briefly stated as follows: desquamation of the ciliated epithelium, edema and swelling of the submucosa, and, in the severer grades, infiltration of the latter with leukocytes.

**Etiology.**—With rare exceptions tracheo-bronchitis is produced by the direct extension of a catarrhal inflammation from the nares, pharynx, and larynx. Rarely the bronchi are the seat of primary acute catarrh, and in some of the latter instances the upper air-passages are implicated secondarily, constituting a reversal of the usual direction of extension above referred to.

The immediate causes are mechanical, chemical, and biological irritants, which act directly upon the tracheo-bronchial mucosa; and that bronchitis is frequently due to infection at a time when the resisting power of the system is reduced there can be little doubt. The circumstances disposing to bronchitis are numerous, those pertaining to the individual being—(1) Age, the old and very young being most liable; (2) Debility; (3) Occupation, as in certain trades that expose to irritating vapors. Among the external conditions are—(1) Climatic factors, particularly variability of temperature and humidity; (2) Seasons of the year. "Catching cold" often results from exposure during the spring and autumn months, when the meteorological elements, humidity and temperature, exhibit greatest variability. These two conditions depend substantially upon the same factors.

Acute tracheo-bronchitis arises as a secondary condition in a great variety of diseases, as, for example, the exanthemata and other acute infectious diseases. As shown elsewhere, in certain instances among this large class of diseases the bronchitis is dependent upon the primary infectious process, but in many others it is due either to the inhalation of pathogenic irritants from the throat and higher air-passages or to the retention of bronchial secretions that are apt to accumulate and decompose with resulting bronchitis. The accidental inhalation of particles

of food and saliva may also lead to secondary bronchitis, or the condition may be secondary to chronic affections (*e. g.* Bright's disease, chronic valvular disease of the heart).

**Symptoms.**—Bronchitis of the larger tubes, which extends down to about the second division of the bronchi, is spoken of usually as a "cold." In such cases the onset is marked by recurring sensations of chilliness and by coryza, slight sore throat, and hoarseness, while in young and feeble children convulsions may occur early. Mild febrile symptoms may appear, the temperature ranging from 101° to 103° F. (38.3°–39.4° C.), with slight acceleration of the pulse, and there may be aching in the limbs and lumbar region. With the fully-developed attack substernal soreness, sometimes amounting to pain, is experienced, especially on coughing, and the pain may be referred to the intercostal muscles and the line of insertion of the diaphragm. The respirations are increased in frequency, but there is no dyspnea. There may be thoracic oppression and discomfort until the bronchial secretions become free, and there is a cough which is at first dry and hard. It often manifests itself in longer or shorter paroxysms, particularly on lying down and on rising after a full night's sleep. At the end of one, two, or more days the cough is moist and attended with an expectoration which is at first mucoid and scanty, then muco-purulent and free; later still it is sometimes distinctly purulent. With free expectoration comes relief to the patient. Histologically, the sputum consists mainly of pus-corpuscles with large cells, in which may be seen the so-called myelin droplets of Virchow and carbon particles.

**Physical Signs.**—Upon laryngoscopic examination the mucous membrane of the larynx and trachea may be seen to be reddened and covered by more or less secretion.

Inspection and palpation of the chest are negative, except when the finer tubes become implicated or fever is present, in which case the respirations may be observed to be slightly accelerated. In children the increased rapidity of the respirations is more common and reaches a higher degree. Bronchial fremitus may sometimes be felt. Percussion yields negative results, save in very rare instances, in which there occurs a decided accumulation of secretion in the tubes, when there may be found impairment of resonance posteriorly below the scapulæ. Auscultation usually renders audible a harsh respiratory murmur, and less frequently piping, sibilant, and sonorous râles. In the advanced stage large and medium-sized mucous râles are frequently present, but are by no means always discernible. The râles appear in different seats from time to time, and after coughing may altogether disappear, but only to reappear later.

The **prognosis** varies with the previous constitutional state of the individual. In healthy adults, after a period ranging from a few days to two weeks, the fever subsides, but the cough, though less marked, and the expectoration usually continue for a variable length of time. In old persons and in those greatly debilitated, as well as in those of a gouty or tuberculous diathesis, the cases pursue a more protracted course. There is also in these subjects a tendency on the part of the catarrhal process to extend downward until the finer tubes are implicated. Under these circumstances life may be endangered, and even,



rarely, terminated. In the old and very young the bronchial secretions are imperfectly expectorated, hence they gravitate to the most dependent parts, and tend to induce bronchiectasis. In young children this downward extension of the affection, with resulting broncho-pneumonia and areas of collapse in consequence of dilatation and occlusion of the bronchioles by muco-pus, is a not uncommon and serious event.

The **diagnosis** is reached without difficulty through the symptoms (slight fever, cough, and expectoration), the acute course, and the physical signs (harsh respiratory murmur, dry followed by moist râles, heard on both sides of the chest). The recognition of the long list of cases that constitute the secondary forms will be made easily possible by noting the circumstances under which they arise.

**Differential Diagnosis.**—Bronchitis can readily be separated from *pneumonia* and from *pleurisy with effusion* by its history, by its lighter course, and by the different character and general distribution of the physical signs, especially by the absence of the signs of effusion and consolidation.

When *broncho-pneumonia* develops in the course of bronchitis, dyspnea and fever are increased and the general condition becomes much more grave. There are small patches that yield dulness on percussion, and broncho-vesicular breathing with moist râles can be detected on auscultation.

Bronchitis cannot be separated from the early stage of *whooping-cough*, but when the characteristic cough of the latter is heard all doubt vanishes.

The bronchitis of *measles* before the characteristic eruption appears is distinguished by the red spots upon the anterior half-arches of the soft palate.

*Localized tuberculosis of the lung* and *acute miliary tuberculosis* are apt to be confounded with bronchitis. The points of difference have been given in the discussion of the former diseases.

**Treatment.**—Not infrequently a “cold” passes through its several stages without rendering the patient ill enough to cause him to seek the advice of a physician, and there are many instances in which but little treatment is required, apart from the usual household measures and protection against cold and damp. If seen early, while the coryza is present, the attack may often be aborted by the use at bedtime of a Dover’s powder in combination with quinin (gr. iv–viiij—0.259–0.518); this may be seconded by a glass of hot lemonade, with or without a portion of whiskey, and either a hot bath or a mustard foot-bath. The following morning a saline laxative should be taken. To children a mild calomel purge followed by a dose of castor oil may be administered. The patient should be kept in a warm, moist, equable atmosphere—preferably in-doors—and during this period he should take divided doses of quinin for a day or two. If the above mode of treatment fail or if the patient does not come under observation early, the main objects of treatment should be (a) to render the secretions free, and (b) to hasten the expulsion of the sputum after it has been loosened. The first leading indication is to be met by the use of diaphoretics, diuretics, and relaxants. The subjoined formula combines these classes of agents, and will be found to be highly serviceable:



R. Potassii citrat., 3vj (23.3);  
 Liq. ammonii acetat., 3v (148.0);  
 Spt. æth. nit., 3j (30.0);  
 Vini ipecac., 3ss (2.0);  
 Syr. pruni virg., q. s. ad 3viij (236.0).—M.  
 Sig. 3ss (2.0) in water every two hours until the secretions  
 are loosened.

If the temperature in any given case be maintained at a considerable elevation, such as 102°–103° F. (38.8°–39.4° C.) or over, tincture of aconite (Mxvj—1.065) may be added to the above mixture; and if there be present much tickling with distressing cough, due to irritability of the affected mucosa, codein (gr. ij–iij—0.129–0.194) may be added to the same. For the incessant irritative cough which is present in severe forms of catarrh opium alone is really effective. When the above prescription is not productive of free secretion and troublesome cough continues, I employ the following:

R. Ammon. muriat., 3v (20.0);  
 Codeinæ, gr. iv–vj (0.259–0.388);  
 Spt. junip. co., 3ss (16.0);  
 Mist. glycyrrh. comp., 3iiss (80.0);  
 Syr. pruni virg., q. s. ad 3iv (120.0).—M.  
 Sig. 3j (4.0) every two hours.

Apomorphin is also excellent as a soothing relaxant in doses of gr.  $\frac{1}{20}$  to  $\frac{1}{10}$  (0.003 to 0.006) every two hours. Mild counter-irritation by means of mustard-paste, followed by the application of iodin once daily, is also helpful. The patient should keep to his room, in which the atmosphere should be kept moist and of even temperature. The expulsion of the sputum may demand stimulating expectorants, though rarely. It is to be recollected that when the tracheal secretion becomes copious the period of convalescence is usually reached, and stimulating expectorants are then entirely unnecessary. When, on the other hand, the cough is no longer dry, and on auscultation the râles are found to be moist, and whilst, at the same time, the expectoration is expelled with difficulty, or if the bronchitis tends to become chronic, then such stimulating expectorants as senega, squills, and ammonium muriate are to be employed. In cases in which expectoration continues to be too abundant terebene, tar syrup, and oil of sandal are to be resorted to, the choice of the special remedy being governed by the requirements of the individual instance.

Debility and secondary anemia must be speedily overcome by exhibiting quinin, bitter tonics, iron, and arsenic, and a suitable change of air often yields prompt and excellent results in protracted cases. The treatment of the various forms of secondary bronchitis will be considered in their appropriate connections in this work.

Apart from the method above given, of attempting to abort the attack in children, acute bronchitis is in the main to be treated in the same manner as when it occurs in the adult. Opium, however, is to be used very sparingly, and generally in the form of paregoric. If the secretion is abundant and imperfectly raised, it is well to administer an

emetic, such as the wine of ipecac (3ss-j—2.0–4.0), and repeat in ten minutes if necessary. If dyspnea be urgent and cyanosis be marked in the lips and finger-tips, a prompt emetic is then imperative in order to save life. A child suffering from acute bronchitis should be kept in bed until the fever subsides.

The diet during the dry stage should consist of liquid forms of nourishment, which should, for the greater part, be taken hot. After the "cold" has been loosened solid food should be resumed.

#### CHRONIC BRONCHITIS.

**Pathology.**—The lesions of chronic bronchitis manifest considerable variety both as regards their nature and extent. The epithelial layer is, in great part, missing, and sometimes the mucous membrane is quite thin. In consequence the longitudinal elastic fibers appear unduly prominent. The mucous glands and the muscular coat undergo atrophy in long-standing cases, and the bronchial tubes are dilated (*bronchiectasis*).

In another large group of cases the mucosa is irregularly thickened or infiltrated and granular. Small ulcers corresponding to the mucous follicles are common, and almost constantly emphysema develops in consequence of secondary changes in the vesicular structure.

**Etiology.**—Chronic bronchitis may either be primary or secondary. The affection is, however, almost always a secondary one, and, though sometimes the result of repeated attacks of acute bronchitis, it is much more frequently caused by certain chronic complaints and certain diatheses, such as chronic alcoholism, rheumatism, gout, syphilis, pulmonary tuberculosis, pulmonary emphysema, organic valvular affections of the heart, and chronic Bright's disease. The primary form, which is rare, is the result of exposure to wet or cold or to the daily inhalation of some irritant that produces and maintains a low grade of catarrhal inflammation (dust, vapors). When chronic bronchitis follows the acute form, we are often able to detect the operation of some favoring cause, as age, climate, and season. It is most common in the aged, though younger persons occasionally suffer, and it occurs by preference during the cold season, often recurring regularly in the cold and variable weather of autumn, winter, and spring, and disappearing in summer.

**Symptoms.**—The symptoms are similar to those of acute bronchitis, though rather less severe. Pain is rarely present, the patient complaining merely of a feeling of substernal constriction. There may be soreness at the base of the chest if the cough be frequent and severe, and occasionally in the epigastrium as a result of traction of the diaphragm on the ribs. Cough is not a constant accompaniment, however, but is paroxysmal and varies in severity and frequency. The degree of the violence of the paroxysm depends upon two factors—the character of the bronchial secretion and the seat of the catarrhal inflammation. Thus when the expectoration is tenacious and small in quantity, and when the small-sized tubes are affected, cough is most violent. It also varies both with the weather and the season, as is evident from the fact that there is often an absence of cough in summer, while it returns unfailingly with each winter.

The expectoration differs widely in different cases. It is sometimes abundant and sero-mucous in character. On the other hand, there are cases of dry cough in which there is little or no expectoration. As a rule, however, it is rather copious, and either muco-purulent or distinctly purulent in character. Fever is usually absent, though there may rarely occur a slight exacerbation at night. The appetite remains good as a rule; the bodily weight is also well maintained, and the nutrition may manifest little or no impairment.

**Physical Signs.**—On *inspection* we usually note undue enlargement of the thorax, with a decrease in expansile movements that is due to the associated emphysema. The dyspnea observed is due either to the same cause or to associated asthma.

*Percussion* yields a clear or hyperresonant note. Dulness or impaired resonance is sometimes met with, however, during acute exacerbations, and especially over the bases, and is due to congestion and edema (Fox). On *auscultation* rhonchi of various forms and moist râles are heard, their number and size being in proportion to the extent of the swelling of the mucous membrane and the amount and fluidity of the secretory products. The respiratory murmur is somewhat enfeebled, though roughened, and the expiratory sound is prolonged and wheezy.

**Clinical Varieties.**—Special forms, depending largely upon specific causal factors, remain to be described:

1. The commonest variety of chronic bronchitis has been called the "winter cough of the aged," and, as before intimated, is usually accompanied by emphysema and cardiac disease. For this form the gouty diathesis is often responsible. The cough occurs in paroxysms that are most severe at night, and during the early morning hours it is attended with free expectoration of the secretion that has accumulated during the night.

2. *Bronchorrhea*.—In this form there may be an abundant bronchial secretion, composed largely of serum (*bronchorrhœa serosa*), but more frequently perhaps the expectoration is purulent and thin, containing greenish or greenish-yellow masses. It may at times be thick and purulent. Dilatation of the tubes and resulting fetid bronchitis may be developed as secondary conditions.

3. *Fetid Bronchitis*.—In this variety the expectoration emits the characteristic odor of decomposing animal substances. The fetor may indicate gangrene of the lungs, abscesses, bronchiectasis, decomposition of matter within phthisical cavities, or empyema with perforation of the lung. It may, however, occur independently of the above-mentioned conditions, and hence these must be carefully excluded before the diagnosis of true fetid bronchitis is made. In the latter disease the expectoration is usually copious, and on standing separates into three layers, of which the uppermost is composed of frothy mucus, the intermediate of a serous liquid, and the lowest of a thick sediment, that presents a granular appearance and is made up chiefly of small yellow masses—the so-called Dittrich's plugs. These plugs are characteristic of fetid bronchitis, and are the cause of the fetor. On microscopic examination the Dittrich's plugs are seen to be composed of micro-organisms, chief among which is the *Leptothrix pulmonalis*; they may also contain pus-corpuscles, fat-granules, and crystals of margaric acid.



The condition may be a grave one, and associated with it may be observed ulceration of the bronchial tubes, with dilatation, pneumonia, abscess, gangrene, and rarely metastatic cerebral abscesses. When putrefactive changes take place in the bronchial secretion in the course of chronic bronchitis a new group of symptoms, as a rule, immediately appears. This comprises rigors that occur at irregular intervals and are associated with high fever and increased prostration. Cough and pain in the chest also become aggravated, but these acute symptoms may shortly subside and the usual course of chronic bronchitis be resumed. Even under the latter conditions fetor of the breath and sputum may persist.

4. *Dry Catarrh*.—The cough is both severe and paroxysmal, and there is little or no expectoration. When expectoration is present the sputum is very tenacious and is expelled with great difficulty. An asthmatic disposition is sometimes noticeable in this variety, and emphysema is commonly associated. The dry condition of the bronchial mucosa is evidenced by sibilant and sonorous râles. This form occurs in old persons, as a rule.

5. Osler has described a form of chronic bronchitis that occurs most frequently in women, and dates its onset from a comparatively early period of life. It does not undermine the general health. The cough is most pronounced in the morning, and is accompanied by a relatively small amount of muco-purulent expectoration (3iv-vj—120.0—178.0 daily). An examination of the chest yields negative results. The etiology is as yet uncertain, although the condition seems to proceed from a gouty or tuberculous diathesis in some instances. I have had under observation for several years a young woman, now aged twenty-eight, who has from time to time during the last five years suffered from eczema of the face, and in the intervals, when not afflicted with this disease, has manifested the symptoms of the form of chronic bronchitis under discussion. She comes of arthritic stock.

**Diagnosis.**—The diagnosis of chronic bronchitis is rarely difficult. Since it is usually a secondary condition, it is of the utmost importance to determine the nature of the primary affection. An examination of the heart and of the urine should not be overlooked.

*Pulmonary tuberculosis* is to be discriminated from chronic bronchitis, and the distinctive points are—(1) A clear tuberculous history. In phthisis there are fever and loss of flesh and strength, while in chronic bronchitis fever is absent and the general health is not impaired. (2) In pulmonary tuberculosis the signs of localized consolidation (usually at one or other apex) appear early, while in chronic bronchitis the vesicular structure is not involved. (3) In phthisis the sputum, when examined microscopically, shows the presence of the tubercle bacillus.

In *acute pulmonary tuberculosis* the fever, dyspnea, cyanosis, and increased prostration constitute a group of features that should serve to avert the danger of its being confounded with chronic bronchitis. Co-existing *pulmonary emphysema* is to be recognized by the characteristic symptoms and signs of this complaint. *Primary fetid bronchitis* must be differentiated, as must also the other conditions previously mentioned, in which the breath as well as the sputum may emit the characteristic fetor. In *abscess* of the lung the sputum contains shreds of lung-tissue, including elastic fibers, crystals of hematin, cholesterolin, and

amorphous blood-pigment; usually localized dulness and broncho-cavernous breathing coexist. In *gangrene* there are contained in the sputum shreds of lung-tissue, but separate elastic fibers are often absent, on account of the presence of a ferment that causes a solution of the elastic tissue (v. Jaksch). *Bronchiectasis* is usually unilateral, and gives rise to areas of dulness and other physical signs that are confined to limited areas, while in chronic bronchitis the signs are general.

**Prognosis.**—Recovery is the exception, though improvement may frequently be observed. The course of chronic bronchitis is exceedingly protracted, and the danger from the late development of certain complications and sequels, such as emphysema or right-sided cardiac disease, must be constantly borne in mind. Since the disease is generally a secondary affection, the prognosis in most instances depends upon the outlook in the primary disease.

**Treatment.**—The treatment falls naturally under two main heads—(1) Hygienic, and (2) Medicinal.

1. *Hygienic.*—This has reference, frequently, to the removal of various noxious influences. When the patient cannot make a suitable change of air during the cold season, he must keep his room during inclement weather; he should, however, be allowed to spend as much time as possible in the open air during clear and pleasant weather. The vitiated atmosphere of saloons or public halls is to be avoided. The patient should be carefully clad; he should wear flannels next the skin during all seasons of the year, but his outer clothing need not be unusually cumbersome. If the case be of an aggravated type and the circumstances of the patient permit, he should be sent to a warm latitude in the autumn, in order thus to escape the effects of a severe northern winter. It is an excellent rule to send patients in whom the bronchial secretions are abundant to a dry, warm climate or to a region whose atmosphere is impregnated with the balsamic vapors of the pine. On the other hand, patients with dry bronchial catarrh are most relieved by an equable, moist, warm climate. Among suitable resorts, those that should be mentioned are the Riviera, Cannes, San Remo, Sicily, and Algiers abroad, and Florida, Southern Georgia, and Southern California at home. Change of air becomes not only a means of relief, but also an effective means of prevention if resorted to at the proper time.

Prophylaxis also includes the removal of any diseased conditions that are causally related. The coexistence of cardiac disease, the gouty diathesis, albuminuria, etc. call for the primary treatment of these conditions.

The diet should be generous, but not stimulating, and articles easy of digestion should be selected. Wines and liquors are to be avoided unless special indications for their use exist. Special conditions, however (*e. g.* albuminuria), may render necessary a special dietary.

2. *Medicinal.*—In this disease medicines are palliative in their effects rather than curative. Relaxing expectorants are to be avoided, owing to their depressing action, and the stimulating expectorants are, in a majority of cases, not only valueless, but hurtful, since they are liable to lessen the appetite and disorder the digestion. When, however, the sputum is muco-purulent in character and is dislodged with difficulty, expectorants of this class (squills, senega, ammonium muriate) may be

tried. I have obtained good results from the use of the following in cases attended with severe paroxysms of cough:

R̄. Ol. eucalypti,                    ʒjss-ʒij (6.0-12.0);  
       Codeinæ,                        gr. vj    (0.388).  
       M. et ft. capsulæ No. xvij.

Sig. One every four hours, as required.

Occasionally potassium iodid exerts a curative influence, but its use may be limited to cases that are due to the syphilitic, rheumatic, and gouty diatheses. Five or ten grains of the iodid four times daily may be exhibited, and should there be present a syphilitic taint the remedy should be pushed to the limit of tolerance. The balsam of copaiba is sometimes efficacious, several instances in my own experience having yielded to the following combination:

R̄. Balsami copaibæ,                ʒj-ʒij (4.0-8.0);  
       Ammon. muriat.,               ʒij    (8.0);  
       Extr. glycyrrh. pulv.,        ʒj    (4.0).  
       Mist. ammoniaci, q. s. ad fʒij (96.0).—M.

Sig. ʒij (8.0) every four hours.

Other remedies that possess great value in certain cases are creasote (in ascending doses), turpentine, terebene, tar, the balsams of tolu and Peru, and sandal-wood. H. C. Wood praises sulphuretted hydrogen in cases in which there is profuse expectoration: "From two to four ounces of the saturated watery solution may be administered by the mouth four or five times a day or until the breath has a perceptible odor."

If the vital powers are poor, bitter tonics, as iron, quinin, and strychnin, and other measures calculated to invigorate the system, are indicated. When the sputum is excessive in amount, astringents (zinc sulphate and oxid) are sometimes useful. Astringents may also be used with advantage in the form of a spray when the expectoration is too free. On the other hand, sprays from properly selected solutions (*e. g.* ammonium muriate, gr. v-x ad ʒj—0.324-0.648 ad 32.0) are valuable in assisting expectoration. In fetid bronchitis sprays of antiseptic solutions are to be used, and the following will be found serviceable:

R̄. Acidi carbolic,                    gr. ij-iv (0.129-0.259);  
       Olei eucalypti,                m̄ij-iv    (0.133-0.266);  
       Aquæ,                            ʒj    (32.0).

Sig. To be inhaled from a steam- or hand-atomizer three or four times daily.

Pneumato-therapy has given brilliant results in certain instances, and more particularly in those of asthma and emphysema.

## BRONCHIECTASIS.

**Definition.**—The universal or circumscribed dilatation of the bronchial tubes.

**Pathology.**—Two main forms are recognized—the cylindrical or simple, and the saccular, and both of these may be met with in the same



lung. Rarely the condition is congenital. It may be general or partial, the former variety being always unilateral, the latter sometimes bilateral. In *universal bronchiectasis* the bronchial tubes, throughout their extent, are the seat of numerous sacculi communicating with one another. These present smooth, shining walls, except in the most dependent parts, where ulcers are sometimes seen. Extreme conditions of dilatation may take the form of huge cysts, which may extend to the periphery of the lung; the lung-tissue lying between the sacculi then becomes cirrhotic as a rule. In *partial dilatation* the bronchial mucous membrane is implicated, with an occasional narrowing of the lumen. Most commonly these narrowings are cylindrical, though they may be saccular, and rarely fusiform. The partial is much more common than the general variety.

**Histology.**—When the walls of the larger dilatations are examined microscopically, the cylindrical epithelium is seen to be replaced by a pavement epithelium. The elastic and muscular layers are thin, and the fibers are usually separated. Contained in these dilatations are frequently found secretions that may be fetid.

**Etiology.**—The manner in which bronchiectasis is produced may vary, though in the majority of instances the condition doubtless arises from an involvement of the bronchial mucosa that extends to the sub-mucous tissues and leads to muscular, fibrous, and cartilaginous atrophy. These changes render the wall of the tube unable to resist the pressure of the air in violent paroxysms of cough, and, once the process of dilatation is commenced, the accumulated secretions tend by their weight to further distend the already weakened walls. Thus the elasticity of the latter is impaired, and finally destroyed. The etiological factors show the affection to be secondary as a rule, and are—(1) Chronic bronchitis and emphysema, chronic phthisis (usually when the seat of the dilatation is at the apex), broncho-pneumonia (in children), and compression of a bronchus by a solid tumor or aneurysm. (2) Great thickening of the pleura, especially when associated with bronchitis or interstitial pneumonia, with contraction of the lung. (3) Rarely it is a congenital lesion, and is then usually unilateral.

Among predisposing conditions are—(a) *Age*, bronchiectasis being most common in adult or middle life; and (b) *Sex*, it being more common in males than females.

**Symptoms.**—There is always cough, and this usually occurs in prolonged and severe paroxysms. The attacks take place most generally after the dilated tubes fill in the morning, and a change of posture may excite them. Accompanying the cough there is profuse expectoration, which may amount to a pint or more in twenty-four hours. The sputum is grayish-brown in color and muco-purulent, emitting a sour or, more frequently, a horribly fetid odor. On standing, the expectoration separates into three strata—the uppermost, of brownish froth; the middle, of a thin, sero-mucous fluid; and a thick sediment, of cells and granular debris. Examined microscopically, the sputum is seen to be composed chiefly of pus-corpuscles, with which are intermingled Charcot-Leyden and fatty-acid crystals, the latter being arranged in the form of bundles; also leptothrices, vibrios, and bacteria are found. Elastic fibers may be observed if ulcers be present.

Dyspnea is noted, but is not a prominent symptom, unless some other chronic affections of the chest already coexist or some complication arises. Hemoptysis occurs rarely, and may be due to the bronchiectatic lesion.

**Physical Signs.**—These differ in character according to the size, situation, and nature of the dilatation, and also according to the condition of the surrounding lung-tissue.

On *inspection* retraction of the chest-wall may be noted when chronic pleurisy and interstitial pneumonia are associated. The tactile fremitus is usually increased, but may rarely be diminished. The *percussion* resonance is impaired or even flat, and on *auscultation* bronchial breathing is heard, with occasional râles that have a metallic quality. A *saccular dilatation* immediately beneath the pleura may give a tympanitic note, and may also give typical cavernous or amphoric respiration. These signs are generally discoverable at the base of one or other lung.

**Diagnosis.**—Simple dilatation of slight degree may exist without appreciable signs, and in other instances the breathing is broncho-vesicular over localized areas, with râles displaying increased metallic quality.

#### SACULAR BRONCHIECTASIS.

History of chronic bronchitis, chronic pleurisy, and interstitial pneumonia, or of foreign body.

Cough is paroxysmal, and sputum characteristic and copious.

Tubercle bacillus absent.

Course longer, with little impairment of the general health.

#### PULMONARY TUBERCULOSIS.

History of cough, hemoptysis, with progressive loss of flesh and strength. Family history.

Cough less paroxysmal. Sputum nummular in the stage of cavity.

Tubercle bacillus present.

Course relatively shorter, powers of the system progressively undermined.

#### Physical Signs.

The condition is persistent, but non-progressive. Usually located near base posteriorly.

Generally progressive, more frequently at one or other apex.

*Circumscribed empyema* with a fistulous connection with the lung may simulate bronchiectasis. There is often in such cases a clear history of an acute illness with a sudden onset, the symptoms pointing to pleural inflammation; or there is a period of gradually increasing ill-health with thoracic oppression and dyspnea, especially on exertion. In either event the patient suddenly expectorates, at irregular intervals, large quantities of purulent matter. *Actinomycosis* may also cause conditions that simulate bronchiectasis. The diagnosis may be made by finding granular particles containing the actinomyces in the sputum.

**Prognosis.**—Apart from certain remote dangers (*e. g.* abscess, gangrene), these cases pursue a favorable but exceedingly protracted course.

**Treatment.**—The lesion being a permanent one, there is no known remedy that will either abridge or influence the course of the affection. Again, since the cough is protracted and attended with profuse expectoration, sedatives and ordinary expectorants are contraindicated. For the fetor, antiseptics are to be employed both topically and internally, and a solution of carbolic acid (1–3 per cent.) or thymol (1:1000) is to be used by inhalation. Internally, terebene (M<sub>v</sub>-x—0.333–0.666) in capsules every four hours is valuable; also creasote in increasing



doses ( $m_j$ —0.066, increasing by  $m_j$  each day, till  $m_{vj}$ —0.399—are taken three times daily) is to be persistently employed.

Should the above methods prove unavailing, intrathoracic injections of disinfectants are often resorted to with gratifying results.

In instances in which the dilatation is situated superficially and is not amenable to therapeutic measures, it may be freely opened and thoroughly drained.

## BRONCHIAL STENOSIS.

**Definition.**—Narrowing of the bronchus, due either to constriction or to compression.

**Pathology and Etiology.**—(a) *Stenosis due to Constriction.*—This form is most frequently occasioned by the presence of foreign bodies; by new growths (polypoid) within the bronchi, or by growths without, extending from the lung to the bronchi, and in the case of the smaller bronchi by swelling of the mucosa. The bronchial walls also sometimes become thickened by inflammatory exudates in certain acute and chronic affections, such as syphilis, tuberculosis, and glanders.

(b) *Stenosis due to Compression.*—Compression of one or more bronchi may be met with in a variety of enlargements involving the organs within the thorax, among which are aneurysm, echinococcus cyst, solid tumors, enlarged glands, mediastinal and pulmonary abscesses, and extensive pleural effusion.

**Symptoms.**—The symptoms do not depend upon the cause of the obstruction, but their extent and character are in proportion to the size of the bronchus affected and the degree of stenosis. Dyspnea is the most conspicuous symptom, and when this is marked the accessory muscles of respiration are brought into active play, and still the proper filling of the lungs with air is not accomplished. Under these circumstances the air in the lungs becomes rarefied, and instead of normal expansion everywhere the lower part of the sternum and the lower ribs are *retracted* on inspiration, and expiration is accomplished only with difficulty. Obstruction of the primary bronchus on either side of the chest would naturally be followed by inspiratory retraction of the inferior part of the chest-wall and intercostal spaces upon the affected side. It is to be recollected that the movements of the larynx are slight in bronchial stenosis, while they are marked in laryngeal obstruction. Cough and expectoration are sometimes present, and febrile development of moderate severity is often noted.

**Physical Signs.**—*Inspection* shows defective respiratory movement upon the side involved. The local tactile fremitus is diminished or absent upon the affected side, owing to the obstruction to the passage of the vibrations of the voice to the pulmonary periphery. The *percussion-note* remains unaltered, though less influenced by forced respiration, and particularly expiration, than in health. Pulmonary atelectasis may occur as a secondary event, and is shown by dulness on percussion. The *auscultatory* signs consist of a greatly diminished vesicular murmur on inspiration, due to the diminished amount of air entering the air-



cells during inspiration, and the presence of râles, sibilant and sonorous in character, at the seat of obstruction. Obstruction of a small bronchus may, however, be present without appreciable physical signs, owing to the fact that the surrounding lung-tissue may take on compensatory emphysema.

**Diagnosis.**—The nature and site of the affection may be determined by auscultation, and sibilant and sonorous râles will be conspicuous at the point of constriction. A clear history, together with a careful investigation of antecedent affections of the thoracic organs leading up to the stenosis, are factors that must furnish the etiological data in individual cases after the exclusion of foreign bodies as the possible cause. Tracheal or laryngeal stenosis may be eliminated by careful laryngoscopic examination.

**Prognosis.**—The duration is indefinite, though usually protracted, and most cases yield an unfavorable prognosis. In those instances, however, in which the narrowing is due to foreign bodies the latter may rarely be dislodged and fortunately ejected, thus averting danger to life.

**Treatment.**—The treatment must be addressed to the cause in individual cases. Obviously, the question of the removal of foreign bodies from the bronchi falls within the domain of surgery, though the administration of an emetic has been followed by complete success in certain instances. Obstruction due to stenosis of a main bronchus may be treated by dilatation with bougies, the treatment of course being carried out by a specialist.

## ASTHMA.

(*Bronchial Asthma.*)

**Definition.**—A chronic affection, characterized mainly by paroxysmal dyspnea, due to contraction of the muscles of the smaller bronchi. The paroxysmal dyspnea produced by arterial contraction is also termed asthma by many writers.

**Pathology.**—True asthma is a neuropathic disease. In a majority of the cases, however, there is more or less hyperemia of the bronchial mucosa, due to pneumogastric or vasomotor functional disturbances, and also a characteristic exudate of mucin. In a smaller number there may be no lesions whatsoever, and the condition is a pure neurosis, often of reflex origin. Instances that come to autopsy present the morbid changes peculiar to chronic bronchitis, pulmonary emphysema, and right-ventricular hypertrophy with dilatation.

**Etiology.**—There is present either a constitutional peculiarity or a singular susceptibility of the local muscular fibers to spasmodic contraction, both of which are of unknown nature. The exciting factors are very various, but may be grouped under four heads:

(1) **Acute Bronchitis.**—It must not be forgotten, however, that a bronchitis may be set up by the paroxysms. Curschmann has observed also a local croupous inflammation of the smaller bronchioles in some

of his cases, which he describes as *bronchiolitis exfoliativa*, and which seems to have given rise to the seizures in grave cases.

(2) The inhalation of numerous and widely various *irritants*, as chemical vapors, smoke, fog, dust, and emanations from plants or certain animals.

(3) **Reflex Causes.**—The causal connection between chronic inflammations, nasal polypi, and other obstructive affections of the nasal chambers is a subject that is thoroughly appreciated by the specialist. In the same way, gastric disturbances and, as I have observed in a few instances, intestinal irritation are productive of this complaint.

(4) Asthma may be secondary to, and most possibly excited by, cardiac disease, emphysema, gout, rheumatism, syphilis, Bright's disease, emotional excitement, and irritating lesions in the region of the medulla. Possibly, some of the latter affections merely constitute predisposing factors. In this connection it is to be pointed out that individual liability to the disease depends upon the special etiologic factor.

**Predisposing Causes.**—*Heredity* takes first place, and is, when discoverable, well marked; it is noted in about 50 per cent. of all cases. The complaint is about twice as frequent in males as in females, and, if we except hay asthma, it is more prevalent in winter and early spring than during the warm season.

**Clinical History.**—Hyde Salter's collective statistics show that prodromal symptoms appeared in about one-half the instances (in 111 out of 226 cases). They differ widely, but are chiefly nervous in a great proportion of cases, and appear as irritability of temper, either depression or unusual buoyancy of spirits, headache, neuralgia, drowsiness, and vertigo. Abundant diuresis and digestive disturbances tend to appear.

The attack usually comes on in the night during sleep, and at a definite time. It may develop, however, while awake or, again, though rarely, during the day. The onset may be sudden, but perhaps more frequently the patient first experiences a moderate grade of dyspnea and thoracic constriction. This augments with unwonted rapidity, and often attains to an inordinate degree, until the patient feels smothered, sits up, grasps his knees with his hands, or places the palms upon the bed so as to raise the shoulders and thus reinforce the accessory muscles of respiration. If the attack be severe, he rushes to an open window when able to leave his bed, or sits on a chair and places his arms on the back of another chair, so as to fix the shoulders and thus give purchase to the auxiliary muscles of respiration while frantically endeavoring to maintain the act of breathing. The face is pale, anxious, and soon is bedewed with cold perspiration, while the lips, eyelids, and finger-tips are livid, owing to defective oxygenation of the blood. The temperature becomes subnormal and the pulse feeble and rapid. The clinical picture wears an alarming aspect, but in uncomplicated cases death never supervenes.

**Physical Signs.**—*Inspection* shows enlargement of the chest, which in the advanced stage becomes barrel-shaped. The reason for this is the presence of an increased amount of air in the thorax with a total inability to expel it. The respirations are diminished in frequency to 12 or 10 per minute. The natural rhythm is also greatly disturbed, and in-



spiration is seen to be short and gasping, and followed immediately by expiration, which is greatly prolonged. The expansile movement of the chest is very limited, and in inverse ratio to the patient's efforts at breathing. There is lowering of the diaphragm. *Palpation* is negative in its practical results. *Percussion* yields a hyper-resonance; in advanced cases with associated emphysema semi-tympanitic resonance is common. On *auscultation* the inspiration is found to be short and feeble, and the expiration much prolonged and accompanied by a low-toned wheezing sound that may also be audible to onlookers. A great variety of dry râles are heard, chiefly high-pitched, sibilant, and sonorous, that are more marked on expiration than inspiration. They also change their character and situation frequently. At the close of the attack moist râles may be heard, and occasionally, when bronchitis complicates asthma, the moist râles may be combined throughout the paroxysms.

The *duration* of the attack is various, ranging from a few minutes to several hours, though rarely it may endure a week or two, with spontaneous remissions during the day (*e. g.* when chronic bronchitis coexists). Usually it subsides abruptly, with the expectoration of rounded gelatinous masses and, later still, of muco-purulent material. The former, when floated in water, are found to be composed of the so-called Curschmann's spirals (mucous moulds of the smaller tubes), and the spiral character of these small, ball-like pellets may even be detectable with the naked eye. When examined microscopically their spiral structure is evident. Two forms are recognized: (1) Composed of *mucin*, arranged spirally; in its meshes may be observed alveolar cells, many of which have undergone fatty degeneration. (2) A perfectly clear and translucent filament that is most probably composed of transformed mucin and occupies the center of the coiled spiral of mucin. In the early stage of the attack Curschmann's spirals (Fig. 44) are invariably



FIG. 44.—Curschmann's spirals.

present in the expectoration, and in many instances Leyden's octahedral crystals are also visible. For a time the latter were supposed, though erroneously, to excite the paroxysms by means of their irritating character. Similar crystals are found in the semen, as well as in the blood in certain conditions (*e. g.* leukemia). Müller, Fink, Leyden, and others have demonstrated extremely large numbers of eosinophile leukocytes in the



sputum. Fink and Gabritchewski likewise have found a large excess, up to 15 per cent., of eosinophile leukocytes in the blood. V. Noorden and Swerchewski found the same increase, but only at the times of the attacks.

**Diagnosis.**—A clear history, together with the physical signs and a microscopic examination of the sputum, should lead to correct results. The history alone is inadequate to put the physician upon the right track. *Laryngeal affections*, which give rise to spasm of the glottis and dyspnea, are to be eliminated by the alteration of the voice and the aphonia which are usually present, while the characteristic physical signs of asthma are absent. Again, the dyspnea is inspiratory, not expiratory as in asthma.

*Emphysema* may be confounded with asthma, though the frequency with which the two conditions are found conjoined in the same case must be recollected. The distinguishing points will be considered in connection with the former disease.

**Course and Prognosis.**—In mild cases of asthma there may be but one or two nocturnal paroxysms, with entire freedom from cough and dyspnea during the following day. On the other hand, in severe cases there is a repetition of the paroxysms from three to five or six nights. Under these circumstances in the intervals (usually corresponding to the period of day) there are slight wheezing and some cough. In long-standing cases asthma leads constantly to the development of chronic bronchitis and emphysema, and in such these affections are invariably combined. The paroxysmal character of the affection is often partly or wholly lost, the patient rarely being entirely free from asthmatic dyspnea, combined with cough and muco-purulent expectoration. The periodicity of the attacks varies greatly; in some it recurs monthly or at even shorter intervals, and in others only annually.

There is rarely any danger to life, except when the secondary affection is emphysema and its remote consequence is dilatation of the right ventricle; but the percentage of cases in which recovery actually takes place is comparatively small, since the affection may reappear long after the paroxysms have ceased to recur in the usual manner.

**Treatment.**—The indications for treatment are—(1) to cut short the paroxysms, and (2) to prevent a recurrence of subsequent attacks.

(1) To bring relief during the paroxysms we should ascertain the exciting cause, and remove it promptly if possible to do so. In one of my own cases a prolonged paroxysm was cut short by a calomel purge followed by an enema. An overloaded stomach calls for an emetic, and other causal factors are sometimes removable (*e. g.* congestion of the nasal mucosa, dust, animal and vegetable emanations). If the cause is irremovable, the patient should be kept in a large and freely ventilated apartment, and everything that tends to impede respiration must be removed. The choice of posture as affording the greatest relief may usually be left to the patient.

To cut short the paroxysms: The particular mode of treatment that will afford most speedy relief differs widely in different cases, and not infrequently the patient, as the result of experience, is aware of the remedies that are most efficacious for good. As a rule, however, sedative antispasmodics, relaxants, and stimulants are the classes of medicinal

agents from which a careful selection is to be made; and whilst a great variety of these have been employed, I shall content myself by adducing here only the most valuable and their mode of administration. In the hands of some observers a few whiffs of chloroform have proved highly efficacious, but in my own they have produced only momentary good effects; ether is the safer remedy and may be tried in like manner. In a certain proportion of the cases from four to six drops of amyl nitrite thrown upon cotton-wool or a handkerchief, and inhaled, bring speedy and permanent relief. Of stimulants, coffee is the best: immediately upon the appearance of the paroxysm about one pint of strong coffee is to be taken hot (without cream or sugar), and in this way the seizure may sometimes be arrested. Alcohol when given hot and in sufficiently large doses to induce mild intoxication may be found very useful; and by adding to "hot toddy" a dose of spirits of chloroform an efficient combination is the result.

The inhalation of the fumes of niter-paper<sup>1</sup> often gives quick, temporary, and, less frequently, permanent relief. When employed, the atmosphere of the room occupied by the patient must be well filled with the fumes.

Among depressant antispasmodics are belladonna, hyoscyamus, stramonium, and lobelia, and these seem to be of most value when used in the form of cigarettes. The leaves of the plant employed are first steeped in a concentrated solution of potassium nitrate or chlorate, and a trial should be made of different sorts of cigarettes or pastilles (which are similarly prepared), since all cases are not benefited by the same brand. The inhalation of tobacco-smoke is equally beneficial in a limited number of instances.

A large number of cases, despite the use of the measures above indicated, exhibit an obstinate tendency, and for their treatment no remedy bears favorable comparison with morphin, administered hypodermically, for potency and permanency of its beneficial effects. It is best given in full doses (gr.  $\frac{1}{3}$ — $\frac{1}{2}$ —0.0216—0.0324), and may be combined with atropin or cocain. Strychnin also has its warm advocates (Mays and others). The use of opium, oft repeated, has occasionally led to the establishment of the morphin-habit, as in a case that recently came under my observation; hence it must not be used indiscriminately. I have for a number of years been in the habit of supplementing the action of the first dose of morphin with the following formula:

R <sub>x</sub> . Tr. lobeliæ,	3j (4.0);
Tr. nitro-glycerini (1%),	℥xvj (1.06);
Sodii bromid.,	3v (20.0);
Vini ipecac.,	3v (20.0);
Ext. hyoscyami,	gr. viij (0.518);
Elix. simplicis,	q. s. ad 3iv (128.0).—M.

Sig. 3j (4.0) every one or two hours in water.

In the protracted cases of old asthmatics, associated with chronic bronchitis and emphysema, the above mixture may be also employed, though sodium iodid (gr. v—0.324) should be substituted for the bromid, and the same dose should be given at intervals of three or four hours.

<sup>1</sup> Niter-paper is prepared by dipping bibulous paper (filter- or blotting-paper) in a solution of saltpeter.

(2) In order to prevent subsequent attacks: The history of each case should be carefully inquired into during the intervals, with a view to ascertaining whether any of the numerous causes (bronchitis, gastric disorders, dust, emanations from plants) are discoverable; and if so, efforts to remove them should be instituted. A methodical interrogation of the various organs of the body and their functions must be carried out, and the therapeutic or hygienic indications presented by them, if any, must be met judiciously. The nasal passages should be examined by a specialist, and any causal conditions found therein are to be promptly removed. If the affection be a pure neurosis or due to bronchitis, a suitable climate may often be found in which the patient will enjoy complete immunity from asthma. The choice of the locality cannot, however, be determined by any known rules. The patient must travel from place to place until he finds the climate that possesses preventive properties in his particular case. To those who cannot adopt this plan potassium iodid offers the best hope of relief, though its use must be long continued (gr. x-xx—0.648-1.296, three times daily). The systematic use of compressed air in the pneumatic cabinet, and also the inhalation of oxygen, are worthy of trial. The presence of any conditions of ill-health calls for treatment directed to their removal.

There are also certain means of prophylaxis for impending attacks. Thus, if there be premonitory symptoms, the use of such measures as strong coffee or the "hot toddy" above mentioned, Hoffman's anodyne, stramonium or belladonna cigarets, the inhalation of the fumes of niter-paper or of a few drops of amyl nitrite, or the removal of the sources of irritation, may suffice to ward off the attack.

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## FIBRINOUS BRONCHITIS.

(*Plastic Bronchitis; Croupous Bronchitis.*)

**Definition.**—A rare acute or chronic catarrhal affection of the bronchial mucosa, attended with the production of fibrinous casts that are expectorated in severe paroxysms of cough and dyspnea. These casts, when unfolded, are found to be solid moulds of the bronchial tubes from which they come, being shaped like the branches of a tree, and thus proving that a bronchial tube and its subdivisions had been blocked. When the moulds are large or medium-sized they are hollow, and when from the smaller bronchi they are solid.

**Pathology.**—The pathology is but little understood, but in my own studies I have found the composition of these casts to be identical with that of croupous exudates met with elsewhere, though more dense, perhaps, than the latter. Croupous bronchitis is attended with loss of epithelium in the implicated bronchi, as is the case in croupous inflammation wherever it occurs, but the answers to the questions, "Why should the affection be limited to a definite portion of the bronchial tree?" and "Why does it recur from time to time?" are obscure indeed. In fatal cases the lesions of associated or antecedent complaints, such as chronic pleurisy, pneumonia, and pulmonary tuberculosis, have been found.



**Etiology.**—What the irritant is that causes the condition is unknown, though streptococci have been found in the moulds and in the mucosa. Some of the predisposing causes, however, have been recognized, and are—(1) *Sex*: it being about twice as frequent in males as in females. (2) *Age*: though met with at all periods of life, it is relatively more frequent from the twentieth to the fortieth year. (3) *Season*: the seizures are most common in the spring months. (4) *Epidemic influences*: Pichini has described a group of instances that occurred in individuals in the same locality. (5) *Hereditary influence* has been traceable in a few cases. (6) Other affections, as tuberculosis (quite frequently), chronic pleurisy, and certain skin-affections, as herpes, impetigo, and pemphigus, form antecedent and coexistent conditions.

**Symptoms.**—(a) *The acute form* is rare. It begins with rigors and fever that are soon followed by urgent dyspnea and severe paroxysms of cough, which are usually attended, soon or late, by the expulsion of fibrinous casts, and sometimes rather profuse hemorrhage. Abundant expectoration usually causes amelioration of the severer symptoms. On the other hand, urgent dyspnea, oppressiveness, and severe cough, with little expectoration, are grave symptoms, often leading to fatal asphyxia.

(b) *The Chronic Form.*—The paroxysms recur at irregular intervals and are less severe than in the acute form, the interim varying from one week to a year or more. In a case observed by myself the patient has experienced a recurrence once annually (on or about May 1st), commencing three years ago. Other instances are on record in which the paroxysms have occurred at regular though much briefer intervals. The cases usually manifest ordinary bronchitic symptoms, with or without fever at the onset. The cough soon becomes troublesome and is paroxysmal in character. There is expectoration in the form of rounded masses, which, when unravelled, are found to be true moulds of the affected tubes that exhibit a laminated structure. The larger casts (which are of the size of a goose-quill or even larger) may be hollow, while the smaller ones are quite solid. They are of whitish or grayish-white color. When examined microscopically they are seen to consist of a fibrillated base, a few scattered leukocytes and mucous corpuscles, and, rarely, gland- and blood-cells. Occasionally Leyden's crystals and Curschmann's spirals have been found. Not infrequently the sputum is blood-stained, and occasionally there is profuse hemorrhage.

**Physical Signs.**—Owing to the obstruction offered by the casts, there is a diminished amount of air entering the corresponding part of the lung. As a necessary result the tactile fremitus, local expansion, and respiratory murmur are diminished over the affected area. The note on percussion over the uninvolved portions of the lung is clear or hyper-resonant, though if the portions of the lung supplied by the affected tubes collapse, there is dulness on percussion. Dislodgement of the casts is followed by a return of the normal respiratory murmur.

**Diagnosis.**—From ordinary *bronchitis* it is to be distinguished by the presence of the fibrinous casts, which alone are sufficient for a positive diagnosis. The fibrinous moulds met with in diphtheria and pseudo-membranous croup, with extension into the bronchi, must also be eliminated. The history and course of the latter will, as a rule, suffice to make a positive discrimination, but if doubt remain a bacteriological examination

of the membranous casts should be made. If the Klebs-Löffler bacilli are then found, all doubts as to its diphtheritic nature are set at rest.

**Prognosis.**—The prognosis in the acute form is quite grave; the chronic variety, though pursuing an exceedingly long course that ranges from five to fifteen years, rarely terminates fatally.

**Treatment.**—This is to be conducted on the same principles as those in simple acute bronchitis. In the acute form an attempt should be made to soften and separate the casts by the topical application of steam, by inhalation, and alkaline sprays (*e. g.* lime-water). Pilocarpin was employed in one instance under my own observation with apparent good results; it tends to excite free bronchial secretion. Emetics should be resorted to without delay when the signs of cyanosis show themselves.

In the chronic form nothing can be accomplished by treatment, during the intervals between the acute exacerbations, that will tend to obviate a recurrence of the attacks or to mitigate their severity.

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## IV. DISEASES OF THE LUNGS.

### CIRCULATORY DISTURBANCES IN THE LUNGS.

#### CONGESTION OF THE LUNGS.

##### (*Hyperemia of the Lungs.*)

**Definition.**—The surcharge of the pulmonary vessels with blood. Two forms are recognized: (1) Active hyperemia, and (2) Passive hyperemia.

##### ACTIVE HYPEREMIA.

**Pathology.**—The blood-vessels in the bronchial mucosa often appear intensely injected, and the capillaries in the alveolar walls are prominent, while on section a scarlet-colored, frothy liquid flows. The alveolar epithelium may become swollen and granular.

**Etiology.**—Active hyperemia is usually a symptomatic condition, though rarely it may arise as a distinct primary affection. Active congestion of the lungs exists as an associated condition in many pulmonary affections, as pneumonia, pleurisy, bronchitis, and tuberculosis. On the other hand, active congestion of the lungs may be engendered as an independent affection by the inhalation of hot air, highly irritative substances, as well as by violent physical exercise, the ingestion of large amounts of alcohol, and strong mental emotion. Active hyperemia has, however, little clinical significance.

**Symptoms.**—The capacity of the air-cells is diminished; hence the oxygenation of the blood is markedly interfered with. This embarrassment of the function of respiration is compensated for in part by accelerated breathing, there being a degree of dyspnea proportionate to the extent and intensity of the congestion. There is some cough, accompanied by frothy, bloody expectoration.

The **physical signs** are bilateral, as a rule, and are generally confined

to the bases. Palpation shows increased tactile fremitus. The percussion-note is impaired or, rarely, dull, and it is generally exceedingly difficult to determine the pitch of the note, owing to the fact that both sides are usually involved. On the other hand, when the condition is unilateral and not associated with diseases of the opposite side, the impairment is readily appreciated. The vesicular element of the respiratory sounds is diminished, and the bronchial element relatively increased (*broncho-vesicular breathing*). Less frequently there is bronchial breathing.

**Diagnosis.**—In the presence of the etiologic factors the sudden development of dyspnea, cough, and a frothy, bloody expectoration, and in the absence of fever and the physical signs before enumerated, the diagnosis is easy.

**Prognosis.**—Active hyperemia is frequently followed by collateral edema. Its course is brief, and terminates either fatally in a few hours, in perfect recovery in a few days, or in pneumonia. The condition is therefore ominous.

**Treatment.**—Prompt measures must be instituted in order to arrest the active fluxion. The special causal factors must be actively treated, so as to diminish the quantity of blood in the pulmonary vessels; dry and wet cups over the entire seat of congestion must be tried; and in the worst cases venesection is demanded. Following the application of the cups, turpentine stupes, sinapisms, and linseed poultices may be employed. I have observed excellent results from the use of *veratrum viride* combined with saline purgatives. Other cardiac sedatives may also be employed, including nitroglycerin in full doses.

#### PASSIVE HYPEREMIA.

Passive, unlike active, hyperemia is always a secondary condition, and is quite common. Two forms are distinguishable: (a) Mechanical, and (b) Hypostatic.

(a) **Mechanical Hyperemia** (*Brown Induration*).—**Pathology.**—The pulmonary vessels are distended, the lungs as a whole enlarged, and the air-cells crepitate but little, owing in great part to the encroachment upon the air-spaces by the dark venous blood. The lungs are of a reddish-brown color and afford increased resistance to efforts at cutting or tearing. On section the reddish-brown tint rapidly changes to a vivid red, from oxidation of the hemoglobin when exposed to the atmosphere. The process commences at the extreme base, extends upward, and may finally become general. The interstitial connective tissue is increased, and is often edematous, while the epithelial cells of the alveoli show altered blood-pigment, usually in the form of hemosiderin and responding to the usual tests for iron.

**Etiology.**—Mechanical hyperemia results from the obstruction of the return of blood to the left heart, and among special causative conditions are mitral constriction, mitral regurgitation, dilatation of the right ventricle, and certain cerebral injuries and diseases. It may also be a symptom of asphyxia, and rarely it arises from pressure of tumors.

**Symptoms.**—The most marked feature is dyspnea, particularly when secondary to organic cardiac diseases with failure of the right ventricle. Cough is common, and an expectoration of frothy serum or blood (hemop-



tysis) containing pigmented alveolar epithelial cells, is the most characteristic clinical feature.

**Diagnosis.**—With a clear history, in addition to the dyspnea, cough, and the characteristic expectoration, the recognition of passive hyperemia of the lungs is a simple matter. The prognosis and treatment will be considered in connection with the causative affections.

(b) **Hypostatic Hyperemia.**—**Pathology.**—The parts of the lung that are affected are dark in color and the vessels distended with a transudate of blood and serum. In this way the air-cells may become emptied of air (*splenization, hypostatic pneumonia*), and the resulting condition is in most instances to be regarded as a mild grade of lobular pneumonia. This view is confirmed by the fact that the same etiologic conditions that favor the development of hypostatic congestion also favor to an equal extent the development of hypostatic pneumonia.

**Etiology.**—Feeble cardiac action, as in long-continued fevers, debilitating chronic affections, and in old persons, combines with a prolonged dorsal position of the body (gravitation thus favoring its development) in producing the condition. This explains why the condition is found usually at the bases of the lungs, and is most marked posteriorly. It is common for the same reason in carcinoma, tuberculosis, paralysis, chronic rheumatism, typhoid fever, etc.

**Symptoms.**—The symptoms are wholly indefinite; indeed, none may be present. Priory has pointed out that old persons in the incipency of the disease begin to sleep with the mouth open, so as to effect the entrance of more air. Commencing cyanosis may indicate the development of hypostasis, and a careful physical examination of the lower lobes of the lungs will show increased fremitus, slight dulness, diminished vesicular murmur, and, in the higher grades, bronchial breathing, with liquid bubbling râles.

The **prognosis** is based upon the character of the underlying affection.

**Treatment.**—This is an affection in which the treatment of causes alone will suffice, save in instances that are secondary to organic heart-affections, in which prompt bleedings are to be advocated. From a pint to a quart of blood should be taken, and I have seen happy results from the employment of this measure in extreme cases. Tapping the right auricle when the blood refuses to flow from an arm vein has also been successfully accomplished by competent surgeons. The patient's posture must be changed from the dorsal to the lateral, and even ventral, and as soon as possible he should be gotten out of bed.

#### PULMONARY EDEMA.

(*Edema of the Lungs.*)

**Definition.**—An effusion of serous fluid into the air-vesicles and interstitial lung-tissue. Pulmonary edema is scarcely to be regarded as an independent affection, but as a secondary condition, being in most instances associated with pulmonary congestion.

**Pathology.**—It consists of a transudation of serum into the alveolar walls, interstitial connective tissue, and air-cells, and rarely the process is limited to the interstitial tissue. Two forms may, for the sake of convenience, be recognized:

(a) **Collateral Edema** (*Inflammatory Edema*).—This is usually local in character, circumscribing an area of the lung that is affected by pneumonia, abscess, or pulmonary infarction, and is the result of a mild inflammatory process affecting the vessel-walls. When the condition follows hypostatic congestion the terms “hypostatic edema” and “splenization” have been applied.

(b) **General Pulmonary Edema**.—If congestion be not associated, the portions of the lungs involved by this type look pale; when pulmonary congestion or pigmentation of the tissue is present, the lung appears darker than the normal and the serum is blood-tinged. The weight of the lung-tissue, owing to the more or less airless condition of the alveoli, is increased, and yet, though heavier than the normal lung, the affected tissue does not sink in water. To the feel it is boggy, and pits on pressure, while on section a serous or sero-sanguinolent (if congestion be present) fluid of low specific gravity, and poorer in albumin than plasma, flows from the cut surface. Edema is most frequently observed at the bases of the lungs, though it may become general, and as a rule the surface of the pleura is moist; hydrothorax may be present.

The mode of production of pulmonary edema is not definitely known. Increased fluidity of the blood on the one hand, and increased tension in the pulmonary vessels on the other, seem to be influential factors in many cases. The heightened blood-pressure may be in great part due to a failure of cardiac power, and particularly to failure of the left ventricle (Welch). When weakness of the left is out of proportion to the weakness of the right ventricle, we are apt to have the tension in the pulmonary capillaries greatly increased, at least until transudation of serum is induced. Edema also occurs as a result of weakness of the right ventricle alone, possibly in consequence of the resulting stagnation of the pulmonary circulation. Obstruction to the overflow, such as occurs in weakening of the left ventricle, or even obstruction in the aorta, leads to heightened tension and, secondarily, to paralysis of the right ventricle. The third and most important factor entering into the production of pulmonary edema is the increased permeability of the vascular walls, due to morbid changes that are the result of impairment of their nutrition. This usually arises in connection with toxic and infectious diseases, when the blood also exhibits more or less change, as in cachectic states, uremia, general septicemia, or some of the infectious diseases. Local edema also occurs in the neighborhood of inflammatory foci, as in pneumonia.

**Etiology.**—Pulmonary edema is secondary to pneumonia and acute and chronic affections, but not with any degree of constancy; nor is it especially liable to be associated with congestion or with low grades of inflammation of the lungs. Among the diseases of which it forms a terminal condition are—valvular affections of the heart, fatal forms of anemia, acute and chronic Bright’s disease, cerebral lesions (hemorrhage, traumatism), and acute infectious fevers with failure of cardiac power.

**Symptoms.**—In edema of the lungs the air-space is lessened in direct proportion to the amount of serum occupying the alveoli; hence dyspnea is always present and is often a conspicuous symptom. There are cough and bronchorrhea. The sputum is usually abundant and frothy, and is expectorated with difficulty. At times, and especially in the acute forms, it is tenacious and may give rise to alarming laryngeal obstruction.



It is blood-stained if congestion be combined. The condition does not give rise to elevation of temperature, except in the inflammatory type, in which fever is constantly present. The pulse is accelerated and feeble, and cyanosis, particularly in cases of collateral edema, usually appears. The extremities are cool and often livid.

**Physical Signs.**—The reasons adduced to explain the dyspnea likewise render intelligible the physical signs encountered. There is dulness, though rarely complete, over the areas involved; the vesicular murmur is feeble or absent, or there may be broncho-vesicular breathing. Since the bronchioles contain serum, small râles, having a liquid character, are audible with inspiration and at the beginning of the expiration over the seat of the edema. Vocal resonance and tactile fremitus may be present.

The **diagnosis**, with a clear history, is based upon the incomplete dulness that is usually bilateral and most marked at the bases, upon the bubbling râles heard over the corresponding area, and upon the absence of any febrile movement, except the latter be due to some underlying affection. *Hydrothorax* bears some points of resemblance to edema of the lungs, but in this condition the upper level of dulness is movable in consequence of change of position of the patient, as is not the case in edema of the lungs. On the other hand, in the latter affection moist râles are present, while they are absent in hydrothorax. *Broncho-pneumonia* may be mistaken for pulmonary edema, though it has a different mode of onset. It is also accompanied by fever, glairy, tenacious expectoration, and more sharply-localized areas of dulness than appear in edema.

The **prognosis** is governed by the pre-existing condition to which the edema is due. Thus, if secondary to a general dropsy due to renal or cardiac disease, it often destroys life with great rapidity. Inflammatory edema, following lobar pneumonia, is also grave in the extreme.

**Treatment.**—The treatment does not differ materially from that of the associated or causal affections. The conditions on which a pulmonary edema depends must, however, be sedulously treated, and the limitation of the transudation and the direct removal of the serous effusion from the lungs is of great importance. We should not fail to frequently change the position of the patient's body, so as to prevent the gravitation of blood to the dependent portions of the lungs. I have witnessed excellent results from the use of dry cups placed over the thorax, particularly over its posterior and lateral aspects, and renewed at intervals of six to eight hours. The number applied should range from one and a half dozen to three dozen. In aggravated forms that develop quickly prompt venesection is imperatively demanded. This is a measure which, if resorted to at the proper moment, will often rescue the patient from imminent danger. The condition of the heart and kidneys must be carefully investigated, and any indications presented by them for treatment must not go unheeded.

#### HEMOPTYSIS.

(*Broncho-pulmonary Hemorrhage.*)

**Definition.**—An expectoration of blood. Its source may be the bronchial mucous membrane (usually the small bronchi), and less frequently it comes from eroded vessels in lung-cavities or their walls; rarely from the larynx, trachea, and larger bronchi. When from the bronchial tubes the term *bronchorrhagia* should be applied. The source



of the hemorrhage, however, is not always easily demonstrable, even when it has resulted fatally and the lungs are minutely examined.

**Pathology.**—The lesions are often microscopic, and consist for the most part of ruptured capillary blood-vessels, though larger vessels may also become the seat of erosion or rupture. After death the bronchial mucosa is sometimes found to be swollen, bleeds easily, and is of a dark-red color—soon becoming decidedly pale. The lung-tissue proper may look paler than in the sound lung. When hemoptysis occurs in advance of pulmonary tuberculosis the lung-cavity may contain a ruptured aneurysm, or mere ulceration of an exposed vessel may be observed. I have witnessed small, dark-red, dense masses in the air-sacs scattered throughout the lung whence came the hemorrhage. Doubtless these are blood-coagula, which result from the clotting of the blood after the latter has been carried into the alveoli. Various associated lesions may be observed.

**Etiology.**—(1) **Pulmonary Affections.**—(a) *Pulmonary congestion* from whatever source may result in hemoptysis, though the amount of blood lost under these circumstances is usually small. There are many causes that excite congestion of the lungs, some of which reside in adjacent organs, it being common in organic disease of the heart, and particularly in disease of the mitral segments. That form of pulmonary congestion which is associated with other affections of the lungs, as well as primary active congestion due to inhalation of hot air, irritating substances, and violent physical exercise, may also result in hemorrhage. (b) *Hemorrhagic infarction* may lead to slight hemorrhage (*vide* Pulmonary Infarction). (c) *Croupous Pneumonia*.—In this disease hemorrhage is caused by the rupture of the capillaries, and the blood, when expectorated, has undergone a change, becoming rusty-colored. (d) *Pulmonary Tuberculosis*.—This is pre-eminently the most common cause. Hemorrhage may take place early when it originates from a sharply-limited and minute tuberculous focus, and it may also be attributable to congestion. Undoubtedly its exact source is the mucosa of the small bronchi; later it is the direct consequence of the ulceration of an artery or of the rupture of an aneurysmal sac that has its seat in a branch of the pulmonary artery. After the tuberculous cavities have healed or while quiescent, calcareous masses are, from time to time, expectorated, together with more or less blood. (e) *Ulcers of the Larynx, Trachea, or Bronchi*.—Rarely ulcers in adjacent structures erode the larger branches of the pulmonary artery and cause copious and speedily fatal hemorrhages. Osler observed a fatal hemorrhage in a case of chronic bronchitis with emphysema. (f) *Fibrinous bronchitis* induces hemoptysis by rupturing the capillaries in the bronchial mucosa at the time of separation of the bronchial casts. (g) *Carcinoma of the lung* produces frequent expectoration of blood. (h) *Gangrene of the lung*.

(2) **Diseases of Other Organs than the Lung.**—(a) *Affections of the heart* act as a cause, and especially advanced mitral disease when it is due to pulmonary congestion. It not infrequently develops during the stage of adequate compensation. In a preponderating proportion of the latter instances the hemorrhage is slight, but it may be profuse and recur at intervals for many years. (b) *Aneurysm* of the branches of the pulmonary artery and of the arch of the aorta (usually with rupture of its coats) is a rare cause of hemoptysis.

(3) **Certain diseases**, such as *purpura hæmorrhagica*, *scurvy*, *anemia*, *hemophilia*, and *malignant forms of certain acute infectious diseases* (e. g. yellow fever), cause hemoptysis. In this class of cases the hemorrhages are due either to a diseased condition of the vessel-walls or to blood-changes.

(4) **Vicarious hemoptysis** is not uncommon during menstruation or when amenorrhea is present. Unless occurring at the time of the regular menses it is not to be regarded lightly, and is of the same significance as when taking place in the male. I cannot agree with those authors who contend that hemorrhage from the lungs in women is without the same dire significance as in the opposite sex.

(5) **Arthritic (Gouty) Endarteritis**.—According to Sir Andrew Clarke and others, this is a common cause of recurring hemorrhages in aged persons (over fifty years).

**Symptoms**.—Hemoptysis is so commonly a symptom of that most frequent and dread disease, phthisis, as to raise suspicions of the latter in the minds of the patient and physician as soon as it occurs. It is appropriate, therefore, to note, first, the features of hemoptysis when dependent upon pulmonary tuberculosis, and then to point out its clinical peculiarities when due to other conditions.

In **incipient pulmonary tuberculosis** hemoptysis develops suddenly as a rule, a warm, saline taste, lasting but a few moments, generally preceding the expectoration of blood. The blood is coughed up, and the bleeding may last only a few minutes or may continue for days, the sputum being apt to remain blood-stained for a longer interval. The immediate effect of the hemorrhage, however slight, is to alarm the patient, inducing, besides mental agitation, cardiac palpitation and other nervous concomitants. A small hemorrhage is not attended with any other results, but large ones give rise to the symptoms of shock, combined with those of symptomatic anemia. When the hemorrhage is large, blood to the amount of a mouthful may be ejected with each cough, and in these instances the effect of the profuse bleeding is evidenced by such symptoms as vertigo, syncope, cold extremities, excessive pallor, perspiration, and a rapid, small, feeble pulse. This is followed, if the attack does not prove speedily fatal, by considerable restlessness, and later not infrequently by mild delirium and more or less fever.

In comparatively rare instances the same patient has a single hemorrhage; more frequently he has several at shorter or longer intervals. Large or small bleedings may precede by weeks, months, or even years any rational symptoms or physical signs of pulmonary tuberculosis. In such instances latent foci of disease may be assumed to have pre-existed.

In quantity the hemorrhage varies greatly: there may be less than one ounce ejected or it may amount to a pint or more before the bleeding ceases. In advanced cases in which cavities have formed large vessels may become eroded, followed by copious and dangerous hemorrhage. Fatal hemorrhage may take place into a cavity without the occurrence of hemoptysis, as in a case dissected by Osler at the Philadelphia Hospital. The distinctive characters of the blood discharged are mainly as follows: bright color, very frothy (being mixed with air), and not clotted. A rare exception to the rule may be noted in the case of hemorrhage proceeding from a large cavity, the blood pouring forth in a free, dark stream.



*Physical Signs.*—These are, for the most part, negative. Quite commonly moist bronchial râles are audible on auscultation; palpation and percussion should not be practised during nor immediately after the hemoptysis.

**Hemoptysis not Due to Pulmonary Tuberculosis.**—(a) In *affections* of the *mitral* and *aortic valves*, especially in mitral stenosis, hemorrhage from the bronchi is not uncommon, and the way in which these lesions lead to pulmonary congestion is explained in the discussion of Organic Affections of the Heart. During the progress of these cases, hemorrhages often occur at considerable intervals; they may either be slight, lasting only a few months, or quite free, extending over periods of a few days or a week.

(b) As a rule, in the beginning small hemorrhages occur for several weeks from pressure of an *aneurysmal dilatation* upon the bronchial mucosa, or there may be weeping of blood through the exposed layers of fibrin composing the walls of the sac. The bleeding point can be discovered with the laryngoscope, when an aneurysm of the innominate or of the aorta impinges upon the trachea. A large and often quickly fatal hemorrhage occurs from rupture into the respiratory tract.

(c) "*Arthritic hemoptysis*" is undoubtedly associated with gouty, degenerative changes in the terminal blood-vessels of the lung, though no coarse pulmonary lesions are induced by the recurring hemorrhages. Though the hemorrhages may occur at intervals for years, as a rule they finally become arrested, and only rarely lead to a fatal issue. I have never observed this form of hemoptysis occurring independently of chronic bronchitis. In emphysema and chronic bronchitis small hemorrhages may occur, and occasionally coagula in the form of casts are formed in the bronchi and afterward ejected. It is probable that the source of the large bleedings that occur under these circumstances is an ulcer in the bronchial mucosa.

(d) The hemoptysis that is connected with the *menstrual function* is of frequent occurrence. I saw recently a patient in whom free bleeding has occurred at intervals of four weeks for a couple of years, with an absence of the menses during the same period of time. In another instance, a patient of Dr. Byers, recurring hemorrhages of the lungs took place instead of the regular menstrual discharge for three successive months, and a comparatively rapid form of phthisis was developed. This class of cases must be regarded as grave.

(e) The preceding group is to be distinguished from those cases in which *trivial bronchial hemorrhages* sometimes occur and in delicate, hysterical females. Though these bleedings are accompanied by cough, it is not uncommon to find, upon careful examination, that the blood comes from the upper air-passages.

(f) Hemoptysis may result from *severe injuries* inflicted upon the thorax, and last for days together.

(g) A person may have a single or many recurring attacks of hemoptysis *without assignable cause*, if we except severe muscular strain or intense mental excitement. Although pulmonary tuberculosis does not supervene in instances of this sort, yet not a few may be excited by a permanently limited tuberculous focus which may be indeterminable by the usual methods of examination. I have more than once seen a cure



result from an active course of treatment with creasote and appropriate hygienic measures. In well-marked instances of the kind a complete arrest of the trouble resulted from a change of climate.

**Differential Diagnosis.**—A reliable diagnosis necessitates the certain exclusion of hemorrhage from the higher air-passages, pharynx, esophagus, and stomach. In *epistaxis* the blood may directly enter the naso-pharynx, exciting cough and being discharged as in hemoptysis. A careful examination of the nasal chambers should be made, however, in cases in which the symptoms are suggestive of epistaxis. Bleeding may take place from the gums, from chinks in the pharynx, or from varicose veins. If the seat of the bleeding be the *pharynx*, the hemorrhage is not free, the blood being commingled with a preponderating proportion of mucus; if from the gums, it may be more copious (as in ptyalism or scurvy), and the hemorrhage then simulates that of pulmonary hemoptysis. An inspection of the mouth will disclose whether or not the gums are the source of the hemorrhage.

The distinctive points between hemoptysis and hematemesis will be found in the discussion of the latter affection.

**Prognosis.**—The gravest apprehensions are constantly entertained by sufferers from hemoptysis, but immediately fatal results are of rare occurrence; and of this fact the patient should be repeatedly assured by the attending physician. In case, however, the existence of thoracic aneurysm is definitely known, the consequences of hemoptysis are certainly fatal. With reference to the effect of hemoptysis upon tuberculous pulmonary disease opinions differ widely; I am of the belief, however, that prior to the existence of cavities it exerts a favorable rather than an unfavorable influence upon the course of the disease. On the other hand, in cases in which cavities exist at the time of the occurrence of hemoptysis an opposite effect is observed. The fact that hemoptysis often precedes by prolonged intervals of time the development of pulmonary lesions is an argument in favor of Niemeyer's view, that phthisis is caused by hemoptysis. There can be no doubt, however, that some blood finds its way into the bronchi below the point of bleeding and into the air-cells, setting up in the latter places of irritation and even lobular inflammation. In this way hemorrhages may aid in rendering the tissues more susceptible to tuberculous infection. In cases of profuse hemorrhage, due to aneurysm or to the erosion of large branches of the pulmonary artery in phthisical cavities, death may be suddenly induced, and is caused largely by inundation of the lung and the consequent impossibility of respiration.

**Treatment.**—Since the hemorrhage is ascribable to (1) congestion of the bronchial mucosa, (2) erosion of the vascular walls, and (3) blood-changes, obviously the treatment of individual cases must be modified according to the character of the causal condition.

In many instances of *hemoptysis due to congestion of the bronchial mucosa* the hemorrhages are, comparatively speaking, slight; hence, apart from keeping the patient at absolute rest, little treatment is required. If not excessive, they are often salutary in their effect. If free, the physician's aim should be to decrease the power of the heart's contraction, and to accomplish this end the patient should be placed in bed, and not allowed to change his position or to speak above a whisper. The diet should be light, nutritious, and non-stimulating, all hot drinks

and alcoholics being prohibited. Among cardiac sedatives to be employed with a view to reducing the rapidity of the heart's action and lowering the blood-pressure, if the patient be neither feeble nor anemic, the ice-bag to the precordia is most valuable; if the pulse be full and strong, we may use aconite and other arterial sedatives. Arthur Foxwell<sup>1</sup> recommends venesection in cases in which *venous congestion* is present, and also lays stress upon measures that confine the blood to the systemic circulation—*i. e.* nutritious food, large doses of the nitrites, hot foot-baths, leeches to the anus, and ligatures applied to the thighs and arms. The pulmonary capillaries may also be effectually depleted by the use of salines, which should be given in full doses. In my own experience dry cupping over the chest has been of the greatest service in cases dependent upon pulmonary congestion. Eating ice and partaking freely of iced drinks are also useful measures. If the attack tends to become prolonged and exhausting, we may increase the coagulability of the blood by the use of such remedies as gallic acid, acetate of lead, or calcium chlorid. Hemoptysis is usually accompanied by *cough*, that constantly disturbs the vascular serenity and excites fresh bleeding. For this symptom opium, and in the worst cases morphin hypodermically, should be freely administered.

When hemoptysis is associated with *organic disease of the heart*, the main indication is to strengthen that organ by bodily rest and quiet and by the use of cardiac tonics, especially digitalis. I have had under observation and treatment for several years a young physician who has been suffering from frequent, marked hemoptysis, due to mitral regurgitation, and in whose case the bleedings are readily controlled by the free use of digitalis.

When in *thoracic aneurysm* or *advanced pulmonary tuberculosis* the blood is ejected in mouthfuls, we may safely infer that erosion of the vessel or rupture of the aneurysm has taken place. Here the object is to bring about the formation of a thrombus that will arrest the hemorrhage. Perfect quiet in the horizontal position tends to allay the vascular excitement, and the induction of fainting by venesection is a measure worthy of a trial, though efforts at treatment are unpromising. Opium is contraindicated in the latter class of cases, owing to the fact that if cough be checked inundation of the bronchial system with the blood (the chief danger) will be favored.

In all instances of hemoptysis treatment should not cease with cessation of the hemorrhage. A tendency to recurrence is manifested in many cases, and hence measures calculated to avoid this event must be brought into play. The patient should not be allowed to indulge in a stimulating diet; he should eschew tobacco and alcoholic stimulants, and avoid all physical and mental strain. Every source of bronchial irritation should be carefully avoided, and attacks of bronchitis, however mild, should receive the most careful attention. Moderate exercise is serviceable, as well as a liberal amount of nutritious food.

<sup>1</sup> *British Medical Journal*, 1894, p. 194.



## PNEUMORRHAGIA.

*(Pulmonary Apoplexy.)*

**Definition.**—An escape of blood into the air-cells and interstitial tissue, with or without ulceration of the pulmonary parenchyma.

**Pathology.**—It may be, though rarely, (*a*) diffuse, when the lung-tissue is ulcerated, as in cerebral apoplexy; or it may be (*b*) circumscribed, as when the blood is effused into the air-cells and the interstitial tissue, with rupture of the parenchyma. The latter form will be considered in the discussion of Pulmonary Infarction.

**Etiology.**—*Diffuse pulmonary apoplexy* is caused by the rupture of a thoracic aneurysm that has become adherent to the surface of the lung. Its most common cause is traumatism, especially penetrating wounds of the lung, but adult life and the male sex are to be regarded as predisposing factors. The lung-tissue is sometimes the seat of diffuse hemorrhagic infiltration in septicopyemia and cerebral disease.

**Symptoms.**—These are ill-defined. Profuse hemoptysis, urgent dyspnea, and cyanosis, followed by increased evidences of collapse, together with a clear history, should raise suspicions of the existence of diffuse pneumorrhagia.

The physical signs are indicative of extensive consolidation arising suddenly, and not of the nature of the lesion.

The **prognosis** is practically hopeless, and abscess or gangrene may result if these cases recover from the immediate effects of the hemorrhage.

**Treatment.**—Absolute rest of the body in the horizontal position is the one measure that offers a slight prospect of alleviation, for thus the formation of a clot, followed by arrest of the hemorrhage, is encouraged. It is unwise to use opium to allay the cough, since the action involved assists in ejecting the extravasated blood, which will, in consequence of gravitation and the effect of respiration, submerge speedily so much of the lung-tissue as to hasten the fatal termination. Ergot is not to be given hypodermically, since it raises the blood-pressure in the lesser circulation, but the internal and external use of cold has been highly recommended. With the onset of collapse cardiac stimulants become absolutely necessary, though many cases are so rapidly progressive as to reach a moribund state before remedial agents can be applied by the physician.

## PULMONARY EMBOLISM.

*(Hemorrhagic Infarction; Embolism of the Lungs.)*

**Pathology.**—Embolic infarctions are firm, airless, brown or black, wedge-shaped masses, with their bases usually at the pleura, which soon becomes lustreless and covered with a delicate layer of fibrin. The infarctions may be single or multiple, and sometimes occupy the greater portion of the lobe; in the majority of cases, however, their size equals that of a walnut. Their most frequent seat is at the back of the lower lobe. The microscope shows the presence of leukocytes and red blood-corpuscles in the air-cells and in the alveolar septa. Collateral congestion and edema are frequent concomitants, and, less frequently, pneumonic consolidation appears.



**Etiology.**—The condition is produced by the blocking of the pulmonary arteries by an embolus or thrombus. When the circulation in the pulmonary capillaries is feeble, hemorrhagic infarction may be the result of stasis, and this is probably the most frequent form. It is met with in connection with diseases of the lungs, and also with mitral stenosis or regurgitation. The plug that occludes the blood-vessel may be composed of leukocytes, as in leukocythemia, and the chief sources of the matter that enters into the emboli are the thrombi in the right side of the heart and in the systemic veins. Infectious emboli, resulting in abscess, will be considered in connection with Abscess of the Lungs. Occlusion of a branch of the pulmonary artery cuts off completely the circulation to the territory supplied by that branch, and hemorrhagic infarction occurs as a result, just as elsewhere.

**Symptoms.**—Not all infarctions give rise to symptoms; on the contrary, occlusion of a main branch of the pulmonary artery usually terminates life speedily. The latter accident occurs not infrequently in connection with organic disease of the heart, and if death be not the immediate result or if a smaller branch be occluded, the most alarming symptoms ensue, such as syncope, urgent dyspnea, and convulsions with unconsciousness. The first and most distressing symptom is dyspnea, which is attended by frantic efforts at breathing and by great mental anxiety. Occasionally hemoptysis is an early symptom, and of primary significance if it occur in a patient suffering from mitral disease. If, together with these symptoms, loss of consciousness with convulsions occurs, the diagnosis becomes wellnigh complete. Cough usually supervenes, accompanied by the expectoration of dark, gelatinous, mucoid masses. Large lymph-cells containing blood-corpuscles are found in the sputum, these giant-cells being most commonly seen in instances of organic cardiac affections. They are supposed to transform the blood-corpuscles into pigment-matter.

The **physical signs** may either be negative—as, for example, when the infarctions are small or deeply located—or they may give information as to the seat and extent of the affected part. When present they are the symptoms of sharply-localized consolidation (increased fremitus, resonance, percussion-dulness, moist râles, and bronchial breathing), and it is not improbable that in many cases the physical signs are due, in great part, to associated conditions, such as bronchitis, edema, or collateral consolidation. The appearance of the friction-sound in the course of suspected cases is a great aid in diagnosis. The heart's action becomes enfeebled, the pulse is small and frequent, and the surface of the body is cool and frequently bedewed with cold sweat. Fever may either be present at the onset or absent throughout. The signs of embolic abscesses in the lungs will be elsewhere detailed (*vide* Pulmonary Abscess).

**Diagnosis.**—To establish the diagnosis of pulmonary embolism there must be a clear history of some etiologic condition, and the sudden appearance of such symptoms as dyspnea, cough, bloody expectoration (in particular), chest-pain, loss of consciousness, and convulsions, corroborated by the physical signs of a sharply-defined spot or spots of consolidation.

**Prognosis.**—The prognosis differs with the character of the primary condition. On the whole, it is exceedingly grave, though the absorption of an embolism, followed by the disappearance of the urgent symptoms,

is not impossible. In case death does not occur soon, infarcts may give rise to abscess or gangrene, the result either of the presence of bacteria in an original embolus or of their entrance through the air-passages. In other cases an infarct may undergo fibroid change and contraction, and may even calcify.

**Treatment.**—Beyond procuring absolute rest of the body and a relief from the distressing symptoms, the treatment should be aimed at the affections on which this form of embolism depends. Dyspnea and pain may require the hypodermic use of atropin and morphin, preferably in combination.

## CHRONIC INTERSTITIAL PNEUMONIA.

(*Fibroid Induration ; Cirrhosis of the Lung.*)

**Definition.**—A chronic exudative inflammation of the lungs, characterized by the formation of fibrous connective tissue. It may occur as a primary or as a secondary affection.

**Pathology.**—Two leading forms of cirrhosis of the lung may be recognized: (*a*) *Local*, and (*b*) *Diffuse*, though these do not demand separate description. It is a unilateral affection, and the lung of the side involved is much shrunken, its dimensions in some cases being incredibly small. I have seen one instance in which the organ measured four inches in its longest and less than three in its shortest diameter. It lies tightly against the spine, and has frequently been overlooked, the heart occupying the affected side, being drawn in that direction during the progress of the disease. The heart is enlarged, chiefly due to hypertrophy of the right ventricle, and the pulmonary artery is the seat of atheromatous change. The other lung is overdistended (*compensatory emphysema*), and may encroach upon the mediastinum. Intrapleural and pleuro-pericardial adhesions may be exceedingly firm and thick on the one hand, and only moderately so on the other, though rarely the pleuræ are intact. The cut surface of the affected lung is hard, dry, airless, shiny, and usually light-gray in color (rarely, reddish-yellow), and the lung-tissue cuts with great resistance. The mouths of the blood-vessels and bronchi, which are often greatly dilated (bronchiectatic), may be observed gaping in the cut section. Cavities may be wholly or in part due to the superaddition of a tuberculous process, though even when the affection is non-tuberculous they may be quite numerous. Phthisical cavities may often be discriminated by their usual situation at the extreme apex. The lung that is unaffected by the fibroid process is also quite often the seat of tuberculous change.

**Etiology.**—The disease is almost invariably secondary, and very generally accompanies prolonged inflammatory and chiefly local changes in the lungs. It may also follow acute inflammatory processes. Examples of **localized interstitial pneumonia** are seen in connection with pulmonary tuberculosis, emphysema, syphilis, hydatids, and fibroid induration secondary to thickening of the pleura.

**Diffuse interstitial pneumonia** has a variety of causes: (*a*) It may follow *acute lobar pneumonia* in cases in which resolution is delayed, and



here the fibrinous exudate filling the air-cells becomes organized into connective tissue. Fibrous tissue is also substituted for the alveolar walls. The condition is exceedingly rare, and no instance of the sort has fallen under my own observation.

(b) Pneumonia, appearing as a complication in influenza, is very liable to produce chronic interstitial pneumonia.

(c) The disease may also result from atelectasis due to compression, as by aneurysms or neoplasms.

(d) It most frequently, however, follows *broncho-pneumonia* in either its acute or subacute form (Charcot). The process starts in the bronchi and extends to the surrounding lung-tissue, till finally an entire lobe, or even an entire lung, may become involved. Tuberculous broncho-pneumonia also leads to the production of new fibrous tissue, but here the process is a conservative one (*vide* Pulmonary Tuberculosis), and hence is not to be classed with chronic interstitial pneumonia.

(e) The initial lesions may be located in the *pleura*, and the lung become involved as a sequel, and the chief lesions may be located in the adherent pleural membrane, with bands of connective tissue extending into the lung. The bronchi are inflamed and sometimes dilated.

Chronic interstitial pneumonia may, however, exist without implication of the pleura, and in view of this fact the primacy of pleural thickenings cannot be granted unqualifiedly when they form a part of the lesions of fibroid induration.

The various forms of the disease thus far described arise *secondarily*. It may also occasionally originate as a *primary* affection (1) from the inhalation of different forms of dust (*vide* Pneumonokoniosis). (2) Delafield describes "a special form of lobar pneumonia." He contends that lobar pneumonia terminates only in resolution or in death, and that this special disease, with its production of newly-formed connective tissue, is from the first a special form of inflammation of the lung. This variety runs a subacute or even chronic course, and terminates by crisis. It is an exudative inflammation, with the formation of new tissue from the onset; but the consolidated areas are not so large as in ordinary pneumonia, and cut sections lack the granular character of the latter.

**Symptoms.**—The patient suffers from cough, which increases in intensity with the progress of the affection. There is a mucous, sero-mucous, or rarely bloody expectoration; dyspnea occurs early, and frequently is present only on ascending heights; and uneasiness, or even pain, over the side of the chest involved may be experienced. In cases in which the bronchi become dilated the characteristic symptoms of bronchiectasis are superinduced. The general symptoms consist merely in a loss of flesh and of strength. Fever is altogether absent.

**Physical Signs.**—*Inspection.*—The chest-wall of the affected side is retracted, while the healthy lung is enlarged (*compensatory emphysema*). The spinal column is curved laterally. The affected side is fixed during respiration, and the heart is displaced by traction toward the affected side. If the left lung be involved, the apex-beat will be displaced to the left and slightly upward; if the right, the apex-beat will be observed to the right of the sternum. The ribs approximate, thus obliterating the interspaces, and the shoulder droops over the shrunk chest-wall.

*Palpation.*—The tactile fremitus is usually increased; if the pleura be



much implicated or thickened, however, fremitus may be decreased. Palpation discovers no expansile motion.

*Percussion.*—The percussion-note varies. Dulness is common, owing to consolidation of the lung, but flatness is occasionally met with, and a tympanitic or amphoric note is sometimes elicited over a dilated bronchus.

*Auscultation.*—The breathing is bronchial or more or less sonorous as a rule, and over bronchiectatic cavities it is cavernous or, rarely, amphoric. Near the base it is frequently feeble, distant, and even altogether suppressed. Subcrepitant, sonorous, sibilant, or gurgling râles may be audible, and dry, creaking, or leathery friction-sounds may also be heard.

**Prognosis.**—The course of the complaint is exceedingly chronic, and lasts over many years. Death may result from an intercurrent attack of acute pneumonia affecting the other lung. The disease always shortens the duration of life, and less frequently is the direct cause of death. Rarely a fatal issue is due to dilatation of the right heart, followed by tricuspid regurgitation.

**Treatment.**—The condition is incurable. The patient should, however, be placed under the best sanitary conditions, and if practicable he should make a permanent change of climate. A suitable resort should be selected in accordance with the rules indicated in the treatment of Pulmonary Tuberculosis, and every effort should be put forth to improve the general nutrition of the patient. Due attention should be given to the associated bronchitis, as well as to any symptoms that may arise during acute exacerbations.

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## BRONCHO-PNEUMONIA.

(*Capillary Bronchitis; Catarrhal Pneumonia.*)

**Definition.**—An inflammation of the minute bronchi and air-vesicles, due either to the extension of inflammation from the capillary bronchi to the air-vesicles or to an inflammatory process set up in atelectatic lobules.

**Pathology.**—Macroscopically, the lungs present decided variations in persons who have died of broncho-pneumonia. On the pleural surface may be noticed purplish or slaty patches, often sunken (atelectasis), intermingled with the more elevated patches of healthy lung and grayish consolidation, and smoother and more moist than croupous pneumonia. Similar appearances are presented by the cut surface. On pressure fluid exudes—edematous from the healthier areas, and grayish and puriform from the consolidated areas. The mucosa of the large bronchi may look natural, though frequently it is congested, while the small bronchi usually contain more or less muco-purulent material. Their walls are greatly thickened, and on section the cut surface presents a nodular appearance. Dilatation of the smaller bronchi may be observed, and minute consolidated areas, varying in size from that of a pin's head to that of a pea, may be seen surrounding the thickened walls of the bronchi. When, as frequently happens, they become confluent, large areas—an entire lobe and even an entire lung—of lung-

tissue may become consolidated. The solidified zones are firm to the touch, being destitute of air, and at first they contain blood; hence their color is a dark-red, but later it presents a grayish hue. The condition is usually bilateral. As a rule, the bronchial glands are swollen and inflamed. In the non-consolidated portions of the lung the air-cells are found to be considerably dilated. The pulmonary pleura is often coated with fibrin, but less regularly than in croupous pneumonia.

The essential lesion is a productive inflammation of the bronchi and of the immediately surrounding air-spaces. The inflammation is from the first not exudative, but productive; that is, with the formation of new tissue (Delafield). This form of inflammation naturally lasts for a longer time than would the exudative, and merges into a chronic productive inflammation of the lung, it may be with subsequent sclerosis or chronic thickening of the pleura. The exudate is always more marked toward the center of the process, while the air-cells toward the periphery show much less exudate. The latter consists of serum, some mucus, and many swollen cells from the alveoli (soon showing fatty degeneration), leukocytes, and also red blood-cells in small numbers. Fibrin is seen in small quantity if at all.

In deglutition and aspiration pneumonia the leukocytes are in much larger number, and the exudate tends to suppuration, while in the more hemorrhagic forms the red blood-cells are present in larger numbers.

Kikodse<sup>1</sup> found the blood in broncho-pneumonia to contain an increased number of white corpuscles, except in fatal or very severe cases. The cause of this increase appears to be the return into the circulation of the corpuscles that have passed into the alveolar spaces; hence it ceases after the fever declines.

Among the associated lesions that remain to be mentioned are—(a) Catarrhal inflammation of the mucous membrane of the bronchi; and (b) Exudative inflammation of the air-cells, which become filled with epithelium, fibrin, and pus, with resulting consolidation of the lung. The epithelial cells lining the air-sacs, since they are more numerous in young children than in adults, form a larger part of the inflammatory exudate in the former than in the latter.

**Etiology.**—(1) A marked predisposing influence is *age*, the disease being most prevalent amongst young children. In them it may appear in association with measles, whooping-cough, scarlet fever, and diphtheria, but not infrequently it is entirely independent of these diseases. Infants are especially susceptible to the affection, most instances of pneumonia at this period of life being of the lobular form. Other conditions that act as predisposing factors in children are improper exposure to cold, unsanitary surroundings (especially impure air), rickets, and chronic diarrhea. Broncho-pneumonia is also frequent in the aged, often being occasioned by certain debilitating causes and chronic diseases that are common to advancing years.

(2) *Season.*—The affection prevails especially in the winter and spring months; particularly is this the case in those forms that are unassociated with the acute infectious group of diseases.

(3) It also supervenes as a complication in such acute infectious diseases as influenza, typhoid fever, erysipelas, and small-pox, and is of

<sup>1</sup> *Annual of the Universal Medical Sciences*, 1892, vol. i. sec. A.



serious import. According to my own observations, it is more commonly met with in the diseases above mentioned than is lobar pneumonia.

(4) The *inhalation of food-particles and other substances* often serves to convey the agents of inflammation to the lobules of the lungs. Thus a long-continued recumbent posture disposes the patient to broncho-pneumonia, since it affords a ready entrance to inflammatory irritants. It is, however, in conditions in which the larynx and bronchi have totally or in part lost their sensitiveness—as in coma due to apoplexy, uremia, and allied cerebral states—that retention of bronchial secretions occurs, and that, owing to gravitation, these secretions reach the minute bronchi. Particles of food and drink are also inhaled. *Inhalation pneumonia* may follow operations upon the nose, mouth, larynx (tracheotomy particularly), and is often secondary to carcinoma of the larynx and esophagus. It is also the pneumonia of new-born children.

(5) It must not be forgotten that very frequently broncho-pneumonia is caused by the *tubercle bacillus* (*vide* Pulmonary Tuberculosis). A sub-acute type may also occur in the course of vesicular emphysema.

(6) The work of Weichselbaum has shown the presence of streptococci with the greatest frequency. The pneumococcus has frequently been found, and in a goodly number of cases the staphylococcus aureus (Neumann, Birch-Hirschfeld), while in influenza the specific organism may itself cause broncho-pneumonia (Pfeiffer and others). Numerous other organisms have been found, and it seems a well-established fact that various pathogenic bacteria may cause the disease.

**Symptoms.**—Two clinical forms may be distinguished: (a) primary; and (b) secondary.

(a) **Primary broncho-pneumonia** is met with most frequently in adults, and presents, for the great part, the symptoms of an acute bronchitis of severe grade (cough, dyspnea, pain, fever, and prostration). When occurring in subjects previously enfeebled the onset may be somewhat gradual. The cough is attended with a catarrhal expectoration that is glairy and tenacious, and may be tinted with bright-red blood in the form of droplets or points. The fever is moderate, the temperature ranging from 101° to 104° F. (38.3°–40° C.), and is of irregular type; in severe cases, however, continued high temperature may occur. Physical examination gives the same result as in the secondary form (*vide infra*). The duration is from two to four weeks, the fever terminating by lysis.

(b) **Secondary broncho-pneumonia** is the variety usually met with. The symptoms are frequently veiled by those of the primary affection, and, indeed, a moderate grade of lobular pneumonia is frequently unsuspected during life when arising in the course of other grave diseases.

It is usually preceded by bronchitis affecting the larger bronchi, and in this common event the first symptom that directs attention to the disease is the sudden increase in the frequency of the respirations, which rise as high as 60 or even 80 per minute. An initial chill is rare. Fever develops suddenly, or, if previously present, increases rapidly. An early symptom is the cough, which is usually hard, harassing, frequently painful, and accompanied by expectoration. The pulse-rate is abnormally frequent, and in the later stages may be quite rapid, feeble, and irregular. The type of the fever is similar to that of the primary form.

*Physical Signs.*—At the beginning of the attack the only sign is the



presence of subcrepitant and sibilant râles, pointing to a general capillary bronchitis. Shortly larger or smaller areas of consolidation become manifest. At first rapid breathing, and soon cyanosis, affecting first the lids and conjunctivæ, may be observed; later, the face becomes dusky



FIG. 45.—Illustrating broncho-pneumonia. The dark spots represent the consolidated areas; the white dots indicate râles: A, coalescence of two areas of consolidation.

and the finger-tips blue. Palpation shows defective expansion and increased tactile fremitus over the consolidated areas. The percussion-note is dull or, less frequently, hyperresonant if the area be small. Auscultation reveals numerous fine, subcrepitant râles, corresponding to the consolidated portions. The respiratory murmur may be bronchial, though more often broncho-vesicular. The signs are usually noted in both lungs.

**Duration.**—(1) In children this varies considerably in different cases. Rarely do fatal instances last more than two or three weeks, while they may be as brief as two or three days. On the other hand, cases in which recovery ensues frequently last from six to eight weeks, though in some instances from one to three weeks only. Two special forms demand brief description:

(a) The *cerebral*, in which restlessness, convulsions, and delirium be-

come so marked as to overshadow entirely the pulmonary symptoms. Not infrequently the onset is characterized by convulsions, high fever, prostration, and alternating stupor and delirium. After such symptoms have continued for from two to five days, pulmonary symptoms appear, while the cerebral decline.

(b) Other cases may manifest a *subacute onset*, in which there is anorexia and occasional vomiting, with the nervous symptoms before noted.

(2) The protracted forms are those in which (a) the symptoms of acute broncho-pneumonia give place to those of a similar though chronic state. The general disturbances may not be marked in some instances, but usually there are cough, loss of appetite, or inability to gain in flesh and strength, and the signs of consolidation persist. (b) Those presenting fever of an irregular type, together with decided prostration, in addition to the symptoms of the preceding variety. In many of the latter instances the lesions are tuberculous in nature.

In adolescence the cerebral symptoms are not as well marked as in children. Two anomalous varieties are met with in practice that demand brief separate description :

**General Broncho-pneumonia.**—The attack develops suddenly and is severe. There are chills, high fever, marked prostration, headache, chest and loin pains, a rapid pulse (soon becoming feeble), rapid and labored respirations, cyanosis, restlessness, delirium, and cough that is at first dry, and followed by mucous, muco-purulent, blood-tinged sputum.

The *physical signs* are defective expansion and an increased tactile fremitus. The percussion-note may be either normal, tympanitic, or dull ; the auscultatory signs are large moist, subcrepitant, crepitant, sibilant, and sonorous râles over both lungs, and a harsh or broncho-vesicular respiratory murmur. The affection is very grave.

**Resembling Tuberculous Broncho-pneumonia.**—The symptoms appear slowly, and the case pursues a subacute or even chronic course. Cough, catarrhal expectoration, moderate fever (often of a hectic type), and night-sweats are noted.

*Physical examination* discloses general bronchitis, together with circumscribed areas of consolidated lung-tissue. Resolution may take place at the end of eight or ten weeks, and complete recovery ensue ; when, however, this favorable event does not occur, the case drags on for an indefinite period, and finally terminates fatally. There are no bacilli in the sputum.

**Diagnosis.**—This can be arrived at by considering—

(a) The nature of the antecedent affections and their etiologic circumstances ;

(b) The distribution of the consolidated areas in both lungs ;

(c) The fact that the physical signs of consolidation are subsidiary to those of general bronchitis ;

(d) The intense dyspnea and cyanosis ;

(e) The type of the fever, irregular as a rule, and its gradual decline ;

(f) The frequent long duration.

**Differential Diagnosis.**—Doubtless, *lobar pneumonia* is constantly mistaken for broncho-pneumonia, and particularly when, in the latter disease, a large portion of one or both lungs becomes inflamed in consequence of the coalescence of small areas of consolidation. The points of distinction may be tabulated as follows :

## BRONCHO-PNEUMONIA.

## LOBAR PNEUMONIA.

*Etiology.*

Presence of pathogenic organisms<sup>1</sup> (streptococci).

Usually secondary to bronchitis and acute infectious diseases (*e. g.* measles, whooping-cough).

Presence of the *Diplococcus pneumoniae*.

Usually a primary disease.

*Clinical History.*

Onset gradual.

Fever is, in proportion to the extent of inflammation, of irregular type, and declines by lysis after a variable duration.

Sputum glairy, tenacious, and in adults may be blood-tinged.

Dyspnea and evidence of carbon-dioxid poisoning prominent.

Physical signs of general bronchitis always marked, and usually preponderating over those of consolidation.

Consolidation commonly bilateral.

Duration indefinite, often extending over many weeks.

Consolidated areas liable to become the seat of tuberculous infection.

Onset abrupt; previous health generally good.

Fever is high, of continued type, and falls between the fifth and ninth days by crisis.

Sputum characteristic (rusty or prune-juice).

Respiration panting, but dyspnea and cyanosis relatively less marked.

Signs of bronchitis generally absent, those of lobar consolidation always preponderating.

Commonly unilateral.

Duration definite as a rule, convalescence following crisis.

Far less likely to become the seat of tuberculous infection.

It is also difficult to distinguish *tuberculous broncho-pneumonia* from the disease under consideration. Indeed, a non-tuberculous broncho-pneumonia may be located at the apex of the lung and accurately simulate the symptoms and signs of the tuberculous form. The differentiation is to be based upon the presence or absence of the signs of softening, and upon a microscopic examination of the sputum (which in a child may be vomited). The softening in tuberculous pneumonia does not, however, begin very promptly; but if elastic fibers and tubercle bacilli be found, the diagnosis is at once set at rest.

**Prognosis.**—In broncho-pneumonia the severity and gravity of the symptoms and the extent of the involvement of lung-tissue are proportionate to one another; hence it follows that the disease may either be devoid of serious import or it may be fraught with great danger to life. Its course is subject to decided fluctuations, the periods of exacerbation in the symptoms often marking the time of the development of the gravest features. Apart from the extent of the lung-tissue involved, however, we must consider especially the condition of the patient at the time of invasion. If the constitution has been previously undermined, as is frequently the case in children, broncho-pneumonia is very apt to be fatal. The disease is less dangerous when it develops in the course of, or follows, measles than when secondary to whooping-cough, influenza, or diphtheria. Wiry, thin children seem to stand broncho-pneumonia better than fat, flabby ones (Osler). *Deglutition* and *inspiration* lobular pneumonia, especially when occurring after operations upon the larynx or

<sup>1</sup>The diagnostic value of the discovery of streptococci is not pronounced. Numerous other organisms have been found in broncho-pneumonia in their absence, and a similar organism (*Streptococcus pneumoniae*, Weichselbaum) has been found in a number of cases of croupous pneumonia.



trachea, are frequently fatal. The mortality-rate in this disease varies from 25 to 50 per cent.

**Treatment.—Prophylaxis.**—There are few diseases that can be so effectually prevented as can broncho-pneumonia. In the first place, proper attention to the mouth as well as to the position of the patient (which should be changed frequently) during attacks of acute infectious diseases will prevent its development in a great proportion of this large class of cases. Adequate protection against exposure to cold during convalescence from measles, whooping-cough, etc. is also a potent factor in preventing the disease, as is the timely handling of catarrhal affections of the nose, pharynx, larynx, and larger bronchi.

Certain *sanitary arrangements* are of the utmost practical importance. The sick-room should be well ventilated and its atmosphere kept at a uniform temperature—68° to 70° F. (20°–21.1° C.). The air of the room should also be well laden with moisture, which may be generated from a croup-kettle or other suitable vessel.

**Local Measures.**—In young children the chest should be enveloped in a jacket-poultice of linseed meal, which should be covered with a layer of oiled silk or waxed paper so as to prevent its growing cool. This should be renewed at intervals of about six hours. After the more active symptoms have subsided the linseed jacket-poultice may be replaced by one of absorbent cotton, which should also be covered with oiled silk or waxed paper. In older subjects the application of iced poultices to the chest exercises a most favorable influence, not only upon the local inflammation, but also upon the fever and the nervous system.

**Treatment of the Attack.**—In cases in which there is high fever, tub-baths should be employed, the temperature of the water at first being about 95° F. (35° C.), and then gradually cooled to about 80° F. (26.6° C.). The gradually cooled bath or the cold pack may be used two or three times daily. The effects are to reduce temperature, to promote refreshing sleep, and to improve the character of the respiration. This mode of treatment is especially effective in cases that begin abruptly. In such the tincture of aconite or veratrum viride may be employed temporarily. In cases presenting moderate pyrexia cold spongings, combined with the use of the ice-bag to the head, may suffice. The following fever-mixture may be employed, though it is not to be regarded as a substitute for the cold-water method of treatment, but is merely supplemental to the latter:

Ry. Potassii citrat.,	ʒijss (10.0);
Spts. ammon. aromat.,	fʒij (8.0);
Spts. æther. nitrosi,	fʒss (16.0);
Liq. ammon. acetat.,	fʒiij (96.0);
Glycerini,	q. s. ad fʒiv (128.0).—M.

Sig. ʒj (4.0) every two hours for a child of five years.

In children a mild mercurial purge at the outset is advantageous, and subsequently by the use of salines or glycerin suppositories a daily evacuation of the bowels is to be secured. The bodily strength is to be maintained by careful, methodical feeding, milk, eggs, albumin, and broths being the best forms of food. The milk should be predigested

if there be marked pyrexia, and egg-white may be given in cold water with a small amount of sugar or in the form of egg-lemonade.

The cough is often wellnigh constant and very distressing. Frequently the use of remedies that promote secretion, combined with a small dose of opium, will, under these circumstances, afford relief. A useful formula is the following:

R. Vini antimonii,                    ʒj (4.0);  
     Spts. æth. nit.,                    ʒijss (10.0);  
     Tr. opii camph.,                   ʒijss (10.0);  
     Liq. ammon. acetat., q. s. ad ʒij (64.0).—M.

Sig. ʒj (4.0) every two hours, diluted, for a child of from three to five years.

Dover's powder is also of value in relieving the cough. When the expulsion of the sputum is attended with great difficulty the preparations of ammonium often meet the indications. Of these the muriate is the most effective, but, unfortunately, this is often objected to, and we must then rely upon the carbonate or the aromatic spirits. The bronchi may contain an abundance of secretion that cannot be expelled, despite the use of the above measures. Under these circumstances an emetic may be given, composed of the wine of ipecac (ʒj—4.0), combined with alum (gr. xx to xxx—1.296–1.944), and administered to a child every ten or fifteen minutes until emesis occurs.

Cardiac stimulants must be employed as soon as the pulse shows signs of failure, and alcohol and strychnin are required in serious cases. The preparations of ammonium owe much of their reputation in this disease to their stimulating properties. These agents when boldly used may suffice to re-establish the cardio-pulmonary circulation, but if they fail in this and cyanosis supervenes, oxygen should be used in addition. Sudden heart-exhaustion may occur, associated with mucous râles in the larger bronchi and rapidly increasing cyanosis. In such instances prompt relief is to be afforded if we would save life. Alternating douching with hot and cold water and electricity should be given a trial.

## PULMONARY ATELECTASIS.

(*Collapse of the Lungs ; Compression of the Lungs.*)

. **Definition.**—Atelectasis of the lungs is a condition occasioned by the removal of the air from the air-cells—a state directly the opposite of emphysema. The air disappears largely in consequence of the process of absorption.

**Pathology.**—The affected lung-spots sink in water, being non-crepitant. They also present through the pleura a bluish-red tint, and on cross-section a brownish-red color. The surface of the affected areas is smooth and depressed below the level of the adjacent lung-structure. The bronchi supplying the collapsed parts are frequently occluded by inflammatory products, but in all cases, as shown by Legendre and Bailly, they may be inflated by means of a blowpipe.

Apart from more or less distention of the pulmonary capillaries with blood, there are no histological changes in the atelectatic areas, though they are of firm consistence (splenization, carnification). There can be no longer any doubt as to the entire propriety of the pathological distinction between lobular pneumonia and atelectasis.

**Etiology.**—The condition occurs most frequently in the new-born, and is then due to defective respiration. Thus in children dying soon after birth the lower lobes may be found to be atelectatic. When acquired, however, there are three modes of production: (1) The first step consists in a more or less complete plugging of the smaller bronchi with muco-pus and other products of bronchial inflammation. If complete, air can no longer enter on inspiration, and as the contained air gradually becomes absorbed atelectasis is the natural result. This condition is very commonly associated with broncho-pneumonia, especially in children. (2) A frequent mode of origin is through compression of the lungs, resulting from positive intrathoracic pressure, after the normal contractility of the lung has been overcome. Instances of this may be produced by pleural effusion, hydrothorax, pneumothorax, pericardial effusion, great cardiac hypertrophy, a solid tumor, or an aneurysm of the arch. Not infrequently abdominal tumors, excessive meteorism, and ascites make sufficient upward pressure against the diaphragm to cause compression of the lower lobes of the lungs. (3) Conditions that weaken and obstruct the inspiration may produce this disease, such as certain brain-affections, paralysis of the pneumogastric, and paralysis of the chest-walls. Thoracic deformities may produce pulmonary atelectasis, and in extreme grades of kyphoscoliosis the lung occupying the side corresponding to the convexity of the spinal column is small. Whilst the lung-expansion and the growth of the organ are greatly interfered with, however, and particularly if the condition arises in youth, true atelectasis rarely occurs from this cause, owing to the natural retractility of the lung. Among conditions arising from deformities of the chest is the so-called aplasia of the lungs.

**Symptoms.**—Atelectasis is a secondary condition, and its symptoms are very generally veiled by those of the primary disease. It arises frequently in the course of broncho-pneumonia, but passes unnoticed unless it becomes very extensive. Respiration is carried on by the upper and anterior portions of the lungs, and is increased in frequency and labor. The pulse is small, rapid, and feeble; the skin-surface, especially that of the extremities, is cool.

The form presenting the most typical symptoms is that occurring in the new-born. It is evidenced by shallow, rapid breathing, lividity, cold extremities, a faint whining cry, drowsiness, and sometimes by evidences of motor irritation, such as muscular twitching and convulsions. Congenital anomalies of the circulatory organs are associated.

**Physical Signs.**—When it involves a goodly portion of the lower lobes posteriorly, as frequently happens, there is marked retraction during inspiration over the lower portion of the thorax, due partly to external atmospheric pressure, and partly to the contractile efforts of the diaphragm. Dulness on percussion is revealed, though only when the atelectasis is extensive, and the tactile fremitus, though very various, is



generally decreased. Localized compensatory emphysema may present semi-tympanitic resonance over small areas of collapse.

Auscultation shows a greatly diminished or absent vesicular murmur, and, if the area of collapse be large, bronchial breathing. Among associated sounds is the subcrepitant râle, due to broncho-pneumonia, and, indeed, capillary bronchitis and atelectasis are often combined, there being, moreover, no reliable signs that will separate them clinically.

The *aplasia* of the lung that is produced by spinal curvature (*kyphoscoliosis*) richly deserves brief separate description, owing to its clinical importance. In many instances the chest is more or less twisted on its own axis, shortened in the vertical diameter, and thoroughly fixed. Under these circumstances lung-expansion is impossible, and hence respiration is purely diaphragmatic. In many other patients life may be prolonged for an indefinite period, nothing more being observed than slightly labored breathing. Such persons, however, upon great physical exertion suffer from urgent dyspnea, and the development of an ordinary bronchitis may lead to similar results, and even to speedy death.

The **physical signs** are those of localized emphysema, combined with those of more or less compression of the lungs. There is an extension of the cardiac dulness to the right, and other evidence of right ventricular enlargement, to which may succeed dilatation with the usual clinical events produced by the latter condition. Death is not rarely due to this failure of compensation.

Autopsies have shown the lungs to be small and more or less compressed, some portions being almost airless. Areas of emphysema of the lungs are often associated. The right ventricle may be found to be hypertrophied merely, or dilatation may also have taken place. Congenital atelectasis, by keeping up high pulmonary pressure, may lead to a persistence of the ductus Botalli and of the foramen ovale.

**Diagnosis.**—Atelectasis may be distinguished from *lobar pneumonia* by the absence of an initial rigor, fever, crepitant râles, and the pain of the latter disease, and by the characteristic inspiratory retraction of the lower portions of the chest and the smaller areas of dulness.

*Pleuritic effusion* gives a flat percussion-note, the upper level of which varies with a change in the position of the patient—a sign that is wanting in atelectasis.

**Prognosis.**—When the condition is limited to small areas it is rarely serious, but equally seldom does extensive atelectasis lead to recovery. The outlook depends to some extent upon the nature of the associated affections; thus, when it is secondary to whooping-cough and widespread broncho-pneumonia, it is very fatal. Other diseases that may complicate and increase the gravity of the atelectasis are pleurisy and pulmonary tuberculosis. On the other hand, compensating emphysema often coexists, and is to be regarded as salutary in its effects. When due to *compression* by pyo-pneumothorax, tumors, and the like, the prognosis is especially gloomy.

**Treatment.**—The treatment corresponds with that of the primary disease. *Capillary bronchitis*, which is so apt to be followed by collapse of the lobules, must receive active treatment, and prophylactic measures are of the utmost practical importance. The patient should be instructed to practise full inspiration at regular intervals; he should not be allowed

to lie continuously in the dorsal decubitus, but should be told to change his position frequently. Another measure that may effectually prevent the development of a serious condition is the use of cold shower-baths (*i. e.* a stream of cold water poured over the region of the neck), and this can, in some instances, be depended upon as a curative agency when the condition already exists. Tonics and the judicious use of stimulants, together with a nourishing diet, are invariably required. I have also seen good results follow the inhalation of compressed air.

In *kyphoscoliosis* tepid baths are indicated. The heart-condition demands careful attention, and cardiac stimulants are to be resorted to at the first loss of compensation or when compensation fails to become established.

## EMPHYSEMA.

**Definition.**—In general this term implies the presence of air in the interstitial alveolar tissue. As applied to the lungs, however, it relates to an abnormal dilatation of the alveoli, of which two forms are recognized: (1) Interlobular; and (2) Vesicular.

### INTERLOBULAR EMPHYSEMA.

This is produced by the rupture of the air-cells, the air contained in the lung escaping into the interlobular connective tissue. Among its causes are—(a) Injuries of the lung (usually by a fractured rib) and perforating wounds of the chest; (b) Violent paroxysms of coughing, as in whooping-cough; and rarely defecation, parturition, and hysterical convulsions. When arising in this way its favorite situation is the anterior margin of the upper lobe.

**Pathology.**—In the interlobular septa immediately beneath the pleura air-bubbles are sometimes seen to be arranged in well-defined rows. The pulmonary pleura may become detached, and the air-tumors may then become as large as an English walnut or even of greater size. Unlike the condition in vesicular emphysema, these sacs are freely movable, and the air may find its way from the root of the lung into the mediastinal connective tissue, and thence into the subcutaneous tissue of the neck and the wall of the thorax. Rarely these air-sacs perforate the pleura, setting up pneumothorax, with or without pleuritis.

Interlobular emphysema is sometimes associated with advanced vesicular emphysema.

### VESICULAR EMPHYSEMA.

(*Alveolar Ectasis.*)

**Definition.**—Dilatation or enlargement of the alveoli and infundibular passages.

**Varieties.**—The cases are classified into—(1) Compensating. (2) Hypertrophic, and (3) Atrophic forms.

## COMPENSATING EMPHYSEMA.

This variety is limited to certain parts of the lung, and arises in consequence of pathologic changes in other parts of the same organ that prevent full expansion of the lung on inspiration. Hence a vicarious increase in the volume of the air-cells is observed in circumscribed morbid processes such as occur in pulmonary tuberculosis, lobular pneumonia, cirrhosis, and pleurisy with adhesions (particularly when the latter is situated at the inferior border of the lung). An entire lung, unaffected by the primary disease, may be the seat of compensating emphysema when the causal disease invades the whole or a greater portion of the other lung, as in cirrhosis, extensive pleurisy with effusion, lobar pneumonia, and pyo-pneumothorax. When, however, the latter conditions are confined to a portion of one lung, the remainder of the same organ becomes distended also.

As a rule, this pulmonary change is physiologic and beneficial: only rarely secondary atrophy of the walls of the air-cells develops, when the latter may coalesce.

*Symptoms* are not presented by the lungs in consequence of the changes met with in compensating emphysema. The condition is sometimes recognizable by means of the usual physical signs, but even these are not always to be relied upon. Fortunately, its existence may be safely inferred when there is conclusive evidence of the presence of the local causal diseases (broncho-pneumonia, pulmonary tuberculosis, pleurisy, lobar pneumonia).

## HYPERTROPHIC EMPHYSEMA.

*Nature of Emphysema.*—The symptoms are dependent upon a loss of elasticity in the lungs, and, the latter condition being the result of overstretching, the contractile energy of the lungs is in great part destroyed; hence they become permanently enlarged. Nor do the emphysematous lungs contract when the thorax is opened, as they do ordinarily. We may in some cases account for the loss of elasticity in the lungs by the operation of causes that produce an abnormal degree of stretching, either temporarily or constantly; but under these circumstances emphysema would be developed despite the pre-existence of normal contractility of the lung. In true emphysema, however, which develops at a comparatively early period in life, we may safely assume that the retractile energy is defective (probably a congenital condition), and hence in such cases the action of the usual causal factors will speedily engender over-distention, or emphysema may develop even in the absence of the usual causal factors. In these instances there is probably a quantitative as well as qualitative defect in the elastic-tissue element of the lungs.

*Pathology.*—The thorax is enlarged (barrel-shaped), and upon removing the sternum the lungs are found to completely fill the mediastinum, and do not retract as in health. They present a pale, anemic appearance, and may show dark pigmented patches and streaks, while to the feel they appear soft and feathery, though dry. They readily pit on pressure (a leading characteristic).



Immediately beneath the pleura enlarged air-cells can be distinguished macroscopically, and air-sacs as large as a walnut or even larger may present irregular projections above the lung-surface. Occasionally they may be so far detached as to be pedunculated. At the anterior borders a series of air-blebs, resembling a frog's lung, may be observed. In these situations, as well as near the root of the lung, distention is usually more marked than elsewhere. The pleura is also pale, and in patches the pigment may be entirely absent (*Virchow's albinism*).

Upon microscopic examination it is observed that the dilatation starts in the infundibular and alveolar passages. The septa are partially obliterated, the alveolar walls thinned and, lastly, perforated, while in consequence of these changes the air-cells communicate with one another, and thus form larger or smaller air-sacs. The process is an atrophic one, in which the smaller elastic fibers at first disappear, while the larger become less prominent and often ruptured. After the latter changes have begun the capillaries likewise disappear, and the epithelium of the air-cells undergoes fatty degeneration, though in the larger bullæ a pavement layer is retained. The smooth muscular element may also occasionally be seen to be hypertrophied (*Rindfleisch*). The condition from which the clinical phenomena arise is most probably the loss of the capillary blood-vessel system.

The bronchial mucous membrane is usually the seat of chronic inflammation. It may be roughened and thickened, or the submucous elastic tissue may present prominent longitudinal lines, while the bronchial mucosa is covered with muco-pus. The smaller tubes may be dilated (*bronchiectasis*), and this condition may be associated with hyperplasia of the peribronchial connective tissue.

The diaphragm is lowered and the subjacent viscera correspondingly displaced. The heart is pushed downward and somewhat backward, the right side showing well-marked changes; the cavities are dilated and hypertrophied, due to obliteration of the pulmonary circulation; and in long-standing cases hypertrophy of the left chambers may also develop. The pulmonary artery and its branches may be enlarged and the seat of atheromatous degeneration. The liver, kidneys, and other viscera present the changes that belong to long-continued venous engorgement.

**Etiology.**—The affection is often secondary to, and develops in consequence of, other affections of the lung—notably, *chronic bronchitis* and *whooping-cough*. The dry form of chronic bronchitis, in particular, is apt to generate pulmonary emphysema. Under these circumstances the disease is directly attributable to the mechanical influences to which the alveolar walls are subjected during respiration. This abnormal strain attends inspiration to some extent, but mainly expiration, owing to the obstruction to the egress of the air in the smaller bronchi, with increased *intra-alveolar air-pressure*. The increased tension in the air-cells may be accounted for, partly, by the severe and persistent cough, the air being forced during violent coughing into the upper part of the lungs, forcibly expanding them and causing emphysema.

*Bronchial asthma*, on account of the obstruction of the exit of the air from the lungs, produces during the attacks an acute emphysema that may result finally in a condition of permanent over-distention. Certain occupations, such as blowing wind-instruments, or those that

entail severe muscular strain (*e. g.* blacksmithing), act as predisposing causes, and hence emphysema is of common occurrence among the working classes, and is more common in males than females. The disease is often hereditary, there frequently being several sufferers in the same family. During advanced years the lung-elasticity often diminishes, and as a consequence a disposition to emphysema is engendered. On the other hand, emphysema is not infrequently met with in children, and in such there may be a respite during early adult life, with a recurrence at a later period. An emphysematous tendency also results from congestion of the lungs associated with mitral valvular disease.

**Clinical History.**—In nearly all cases the disease develops insidiously, the symptoms being gradually added to those of the primary affections (chronic bronchitis, asthma, etc.). When due to the occupation of the patient its development is also slow, and not infrequently its origin dates back to childhood or beyond the recollection of the patient. Rarely it may exhibit a more acute development, as, for example, after whooping-cough.

The first symptom is a variable degree of *dyspnea*, and to this may be added temporary *cyanosis* and *cough*. The severity of the dyspnea varies with the degree of distention of the air-cells, even though additionally aggravated by the coexistence of chronic bronchitis, asthma, etc. In moderate emphysema the dyspnea is only apparent on going up stairs, running, walking rapidly, or after a hearty meal; on the other hand, in advanced grades of the affection it is constant, and is intensified by the slightest exertion, even to orthopnea. Speech is interfered with, the patient's utterances taking the form of fragmentary sentences or syllables. The labored breathing is shown particularly in expiration, and, as in asthma, in which the alveolar spaces are acutely distended, so in emphysema the rhythm of the respiration is changed. The inspiration is shortened, and the expiration is greatly prolonged and accompanied by wheezing when chronic bronchitis coexists.

In the later stages cyanosis becomes more marked, and is noticeable in proportion to the loss of compensation and interference with the cardio-pulmonary circulation. It often attains to an extreme degree, and the patient's alarming appearance may be in striking contrast with his apparent degree of comfort. In mild forms the cyanotic tint is confined to the lips, lobes of the ears, and the extremities. Any increase in the degree of dyspnea after exertion results in an increased blueness of the surface.

The *cough* is dependent upon the presence of chronic bronchitis, and the latter disease is frequently found in combination, particularly during the winter. There is also an expectoration that is identical with that of chronic bronchitis, and when this disease reaches an advanced stage the cough persists throughout the year (*vide* Chronic Bronchitis). Intercurrent acute attacks of bronchitis are often followed by temporary attacks of asthma; and since chronic bronchitis in its highest grades is met with at an advanced period of life, so, as would be expected, the cases of advanced emphysema are also met with at the same period. Osler has described a group of cases occurring in patients "from twenty-five to forty years of age who, winter after winter, have had attacks of intense cyanosis in consequence of an aggravated bronchial catarrh."



These patients are short-breathed from infancy, and their condition is attributed to a primary defect of structure in the lung-tissue.

*General Symptoms.*—There is an absence of febrile movement; the pulse is not increased in frequency, though sometimes feeble; and the temperature of the body is generally subnormal. There is a very gradual loss of flesh and strength, and the patient is stoop-shouldered, presenting a peculiar cachectic appearance—a condition that is in strong contrast with the dusky appearance of the face, the swollen neck, and the enlarged chest.

Finally, other symptoms may be mentioned that are for the most part secondary to hypertrophy, followed by dilatation, of the right ventricle. This hypertrophy is the result of pulmonary congestion and obliteration of the pulmonary capillaries induced by the emphysema. Under these circumstances severe attacks of cough occur, attended with extreme dyspnea and lividity, and later the conditions that usually succeed a moderate grade of tricuspid insufficiency supervene, such as congestion of various viscera and edema of the feet. Anasarca is rare.

*Physical Signs.*—The shape of the chest is much altered: owing to the increased antero-posterior diameter, it becomes barrel-shaped (Fig. 46), and the sternum bulges, as do also the costal cartilages.



FIG. 46.—Barrel-shaped chest in emphysema.

The infraclavicular and mammary regions are also prominent, and give the thorax an abnormally rounded appearance. The episternal notch is deeper than the normal, the clavicles and muscles of the neck are unduly prominent, and the neck itself appears to be shortened, owing to the elevated position of the clavicles and the sternum. There is an antero-posterior curvature of the spine and a winged condition of the scapulæ—changes to which the stooping posture is ascribable. Below, the thorax appears contracted. The intercostal spaces are widened and depressed, and a network of dilated venules frequently extends laterally above the inferior costal border, but is by no means characteristic of the affection.

The movements of the chest are vertical rather than expansile, and the lungs are constantly in a state approaching full expansion: in the lower thoracic and upper abdominal regions there may be observed retraction rather than expansion during the act of inspiration. The respiratory acts, as a whole, are labored, and the diaphragm and abdominal muscles are seen working with relative violence. The heart's apex-beat is invisible, but marked epigastric pulsation is frequently



noticeable. A transverse linear depression across the abdomen, on a level with the lower ribs, may also be present during inspiration. Venous pulsation may be seen in the neck after failure of the right ventricle has occurred.

On *palpation* the character and direction of the chest-movements may be accurately appreciated. The tactile fremitus is decreased, but not absent. In advanced cases the apex-beat cannot be felt, and even in the earlier stages it becomes more and more enfeebled. Owing to displacement of the heart and engorgement of the right ventricle there is a distinct systolic shock over the ensiform cartilage, and also a pulsation in the epigastrium.

*Percussion* yields a characteristic hyper-resonance. This may be distinctly "Skodaic" or semi-tympanitic, and in extreme dilatation of the air-cells the tone may be woodeny. The area of cardiac dulness, owing to the fact that the lungs overlap the heart, becomes lessened and finally obliterated; while the upper limit of liver-dulness, both anteriorly and posteriorly, is found to be one or two interspaces lower than normal, owing to the fact that the diaphragm is depressed. The upper level of splenic dulness is also lowered, and the area of percussion-hyper-resonance extends higher above the clavicle than naturally.

On *auscultation* the inspiration is short and feeble, while the expiration is greatly lengthened, the ratio of these sounds as to duration being reversed as compared with the normal. Their pitch is somewhat lowered, particularly that of expiration; and when râles are present the respiratory murmur (particularly the inspiratory) may be scarcely audible. In well-marked instances of emphysema inspiration and expiration may rarely be of equal length. It is a fact worthy of emphasis that the parts of the lungs that are not so markedly emphysematous as others give a harsh, exaggerated vesicular murmur, owing to the great efforts of breathing. Râles of various sorts are frequently audible, due to bronchitis, which, it must be recollected, accompanies emphysema in a majority of instances; less frequently the auscultatory signs of asthma, pleuritis, and phthisis are encountered. Rarely, rubbing sounds, that have been attributed to the friction of enlarged air-cells against the pleura, are audible, and when the interlobular variety supervenes upon vesicular emphysema a *crumpling* sound is heard. The so-called "*Laennec's râle*," which resembles somewhat the subcrepitant râle, is not infrequently present. The vocal resonance varies from an almost total absence to a greatly increased intensity. The tricuspid insufficiency that develops late in this affection is betrayed by its characteristic murmur.

**Diagnosis.**—A positive diagnosis may be arrived at from a consideration of the history, including such points as heredity, occupation, the long duration of the condition, together with the most characteristic symptoms (dyspnea, cyanosis, signs of chronic bronchitis), and from the physical signs. In a case of beginning emphysema, particularly among children, a certain diagnosis is not to be attempted.

**Differential Diagnosis.**—*Pneumothorax* is the disease most apt to be confounded with emphysema. It develops suddenly, however, while emphysema is of slow development, and the rational symptoms of pneumothorax are more constant and urgently distressing than those of

emphysema. Pneumothorax is unilateral, and gives a purely tympanitic percussion-note, while hypertrophic emphysema is bilateral and its percussion-note is hyper-resonant. Auscultation in pneumothorax usually gives amphoric breathing, metallic tinkling, the characteristic succussion splash, and an absence of the vesicular murmur; all of which symptoms are very unlike the auscultatory signs of emphysema.

Another affection giving rise to dyspnea, cough, and cyanosis is *pleurisy with effusion*, but the slow course, the absence of fever, and the universal hyper-resonance that characterize emphysema do not belong to pleurisy. The latter affection is usually unilateral, and over its seat a flat percussion-note is obtained.

**Prognosis.**—Hypertrophic emphysema of acute form (*e. g.* resulting from whooping-cough) is often curable; but the usual slowly-generated variety, so far as recovery is concerned, gives a totally unfavorable prognosis. In many cases, however, life is not materially shortened. Temporary improvement is possible when the lesion consists merely of a distention of the air-cells, and this is shown by a corresponding improvement in the physical signs. The effect of frequently recurring attacks of bronchitis is only to intensify the symptoms of a disease that is innately progressive. Intercurrent affections, however, such as pneumonia (lobar and lobular) and pulmonary tuberculosis, may prove fatal. Dropsy, following broken compensation, is often a late and dangerous complication; other late accidents of the disease are hemoptysis and sudden dilatation of the right heart.

Individual circumstances, such as the patient's social condition, the stage of the affection in which he comes under proper treatment, and the degree of care he is willing to exercise, greatly influence the outcome of the case.

**Treatment.**—The treatment is to be directed toward the removal of the causes of emphysema, and chiefly of the chronic bronchitis. From personal observation I am fully convinced of the fact that the progress of the disease can be arrested, and that the condition is sometimes improved, by relieving the chronic bronchitis. The iodids (potassium, sodium, and ammonium) will sometimes produce effects that are truly remarkable, and the syrup of hydriodic acid may be employed when the iodids are not well borne by the stomach. If the occupation of the patient tends to aggravate the disease, it must be forsaken for one that is less harmful. Violent paroxysms of cough also contribute to the production of alveolar distention, and hence must be alleviated promptly. Intercurrent attacks of asthma have a similar effect, and must be relieved as speedily as possible by a resort to appropriate therapeutic measures. Attacks of acute bronchitis are to be prevented, if possible, by suitable clothing, by avoidance of exposure to inclement weather, dust, and the vitiated atmosphere of overcrowded halls, churches, and the like; whenever practicable the result can be most successfully obtained by a residence in an equable climate. Since a severe bronchitis is apt to increase the severity of the emphysematous symptoms, it must be cured as rapidly as possible.

As soon as passive congestion, flatulence, and constipation, with other gastro-intestinal symptoms, appear, the diet will demand careful regulation, and especially a restriction in the use of carbohydrates.



The bowels must also be moved regularly with a view to obviating the flatulence and portal engorgement.

The heart needs to be carefully watched, and as soon as signs of broken compensation appear digitalis and strychnin will be found highly useful. Diuretics and cathartics may also become necessary. The sudden development of urgent dyspnea (or orthopnea) and extreme lividity, especially if associated with weak cardiac action and a rapid, feeble, irregular pulse, calls for free bleedings, and more than once in the course of my hospital practice have I seen the lives of patients suffering from emphysema saved by timely venesection.

Besides meeting the pathologic and symptomatic indications, we should aim to assist the patient in expiration, and Gerhardt has suggested systematic *mechanical compression* of the thorax during expiration as a useful measure. This external pressure must be made by an attendant, who places his hands flat on the lower lateral portions of the thorax, and the manipulation is to be continued for from ten to fifteen minutes daily. The results obtained by certain German authors have been encouraging, but in my own hands the method has failed, except in two instances occurring in young adults with yielding chest-walls, in whom it was of the greatest service. The *pneumatic treatment*, comprising the inhalation of compressed air and the breathing into rarefied air, richly deserves further trial,<sup>1</sup> its use having been productive of permanent improvement in a number of cases, as shown by physical examination (including mensuration).

#### SENILE EMPHYSEMA.

This variety is in reality a senile atrophy of the lungs, and has been appropriately termed "small-lunged emphysema" by Sir Wm. Jenner. In consequence of the complete atrophy of the alveolar walls, coalition of the air-cells takes place, with the production of large air-sacs. The lungs contain less than the normal volume of air, instead of an abnormal quantity as in true hypertrophic emphysema, and as a result occupy less space in the chest-cavity than do healthy lungs. The pulmonary tissue elements are deeply pigmented. The condition does not produce right ventricular hypertrophy.

The **symptoms** are negative, although subjects in whom senile emphysema develops may have previously had chronic bronchitis with more or less dyspnea. They quite frequently present a withered appearance, and the chest on inspection is seen to be contracted, owing to the fact that the ribs approximate more closely and take a more oblique direction than in health.

**Treatment** is unavailing.

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#### GANGRENE OF THE LUNGS.

**Pathology.**—The affection presents itself in two forms—as a (a) diffuse, and a (b) circumscribed process.

(a) The **diffuse** variety is rare. It may, however, be met with in

<sup>1</sup> Waldenburg's portable apparatus is convenient for use.



lobar pneumonia, and very rarely in consequence of occlusion of the large branch of the pulmonary artery; it may also be secondary to the circumscribed form. The greater part of the lobe, or even an entire lung, may be involved, the pulmonary parenchyma degenerating into a putrid, greenish-black, pulpy mass, with no obvious line of demarcation.

(b) The **circumscribed** form may involve either one or both lungs, though the right is affected somewhat oftener than the left. To this category belongs the so-called *embolic gangrene*, the nodules of which have their favorite seat in close proximity to the pulmonary pleura. All etiologic varieties of the circumscribed form more frequently implicate the lower than the upper lobe of the lung, occurring in sharply defined areas, which may either be single or multiple. The affected area first presents a greenish-brown appearance; its central portion soon undergoes softening, and a cavity is thus formed whose walls are ragged and irregular and contain a foul-smelling, dark, greenish liquid. The surrounding lung is inflamed, and the air-sacs contain inflammatory products (fibrin, epithelium, pus), while the highly-irritating and putrid material sets up an intense bronchitis. These gangrenous foci may increase in size by a peripheral extension, and thus the adjacent veins may become plugged with infectious thrombi or the vessels may become eroded. Emboli may then be detached from the infectious thrombi, and, entering the circulation, may set up foci of septic inflammation in remote organs. A truly remarkable connection exists between circumscribed gangrene of the lung and cerebral abscess. When the gangrenous spot is situated near the pleura, simple or gangrenous pleurisy may arise as a complication, or the pulmonary pleura may be perforated and pyo-pneumothorax result. When recovery ensues the cavities formed as the result of the conversion of lung-tissue present a limiting wall of dense connective tissue. Such cavities may remain permanently or may slowly become contracted.

**Etiology.**—Gangrene of the lungs is caused by the bacteria of putrefaction (probably the staphylococcus albus or aureus). The disease is rare, even though the opportunity for inhaling the bacteria that cause it is a constant one. It is only when the lung-tissue has become impaired or peculiarly altered that the specific bacteria are capable of producing gangrene. It may occur in several ways:

(1) Secondarily to lobar pneumonia, hemorrhagic infarctions, cavities in the lungs, bronchiectasis, wounds of the lung, contusions of the thorax, carcinoma of the esophagus, or to compression or embolism of the pulmonary artery or of the bronchial vessels.

(2) As an embolus, derived from a gangrenous area in some other organ of the body, it may lodge in the lung and set up putrefactive changes.

(3) Pressure from a thoracic aneurysm may give rise to gangrene.

(4) The most important causal factor, however, is the entrance of foreign bodies, especially bits of food, into the bronchi and lungs. Whether or not the specific bacteria of putrefaction enter the lungs with the foreign bodies, the latter render the tissue-soil receptive to the former, and once the process has been initiated it is apt to extend itself. There are several ways in which these foreign particles gain entrance into the bronchi and lungs: (a) By a faulty swallowing of the food; (b)

By inhalation; (c) By a carcinomatous perforation of the esophagus into the bronchus or into the lung.

(5) In the course of debilitated states of the system, as during convalescence from protracted fever (rarely), and in diabetes mellitus (frequently).

**Symptoms.**—These are *local* and *general*, the former alone being diagnostic.

**Local Symptoms.**—There is cough accompanied by an exceedingly fetid expectoration that is usually quite profuse. When abundant, and when expectorated into a conical glass and allowed to stand for a time, it separates into three layers: (a) the uppermost, being frothy, opaque, and of a grayish-yellow color; (b) the middle, clear and watery; and (c) the lowest, appearing as a greenish-brown sedimentary layer containing shreds of lung-tissue and sometimes blood. The microscope shows it to consist of numerous elastic fibers, bacteria, fat-crystals, muco-pus, granular matter, and leptothrices. Small quantities of blood in the sputum are very common. Kannenburg and Streng have also described ciliated monads as occurring in the sputum. The patient's breath is, as a rule, intensely fetid, even though there be no expectoration, but this fetor of breath may be absent, as in a case of my own (which came to autopsy), in which the localized gangrenous process had no fistulous connection with the bronchus. It is to be recollected that if any of the large branches of the pulmonary artery be eroded, free and even fatal hemoptysis will result.

**Physical Signs.**—The physical signs are sometimes obscure, as when the areas involved are smaller and deeply situated, and in such instances signs of bronchitis only may be detectable. When large and favorably situated, however, the affected spots usually give signs of consolidation, rapidly followed by those of cavity. In addition bronchial râles—usually moist—and coarse cavernous râles are usually audible. It is obvious that when the pleura is implicated the signs of pleurisy are added, and if pneumothorax be present those belonging to the latter condition also.

The chief **general symptoms** are irregular fever, emaciation, and profound prostration. A septic condition of the system is commonly developed, and the patient sinks from exhaustion. Rarely there may be an almost total absence of constitutional disturbances, and such instances terminate in recovery.

**Diagnosis.**—The distinctive feature is the intense fetor both of the sputum and the breath. The physical signs may readily determine the existence of the pulmonary lesion, but it is difficult to eliminate *abscess* and *fetid bronchitis* associated with bronchiectasis. The results of a careful examination of the sputum, together with the less horribly fetid odor of the breath, in *abscess* will usually suffice to eliminate the latter affection. In *fetid bronchitis* the fetor of the breath and sputum is also less marked than in gangrene, while its course is slower and more favorable than that of the latter affection.

**Prognosis.**—The prognosis is always grave, though rarely recovery in circumscribed gangrene of the lungs ensues. The chief dangers are exhaustion and hemorrhage. Improved methods of surgical treatment, however, have saved life in a few instances, and promise to reduce still further the mortality-rate of this serious affection.



**Treatment.**—The leading indications are—

(a) The disinfection of the gangrenous focus or foci in the lungs. This may be accomplished by the internal administration of creasote or carbolic acid or by the use of the antiseptic spray. In a recent case the employment of Robinson's inhaler, charged with equal parts of creasote, alcohol, and chloroform, gave encouraging results.

(b) The patient's nutrition must be maintained, if possible, by a concentrated liquid diet, administered in fixed quantities and at regular intervals; also by the judicious cultivation of the digestive functions, together with the use of stimulants and tonics. For a description of the surgical treatment of gangrenous cavities of the lungs the reader is referred to special works on surgery. It is the physician's duty, however, to determine whether or not the patient's general condition will admit of surgical interference, and also to localize as nearly as may be the affected zones for the surgeon's guidance.

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## ABSCESS OF THE LUNGS.

(*Suppurative Pneumonitis.*)

**Pathology.**—This affection is characterized by the formation of pus and the degeneration of lung-tissue. It may be (a) a mere infiltration of the blood-vessels, bronchi, or interstitial tissue, but more frequently purulent inflammation of the lungs takes the form of (b) an ordinary abscess. In size the abscesses range from that of a walnut to an apple, and I have observed in one case inflammation of the whole of the middle lobe of the right lung. The abscess-walls are irregular and decidedly ragged; and in the case of old lesions there is a dense fibrous wall; the contents are purulent and rarely necrotic. If the contour of an abscess touches the pleura, empyema is the usual result, though sero-fibrinous pleurisy may rarely follow. Rupture of the abscess into the pleura may also occur.

**Etiology.**—Streptococci are found, though they are not the only direct causes of abscess of the lung. The diplococcus pneumoniae and Friedländer's bacillus have been noted, as well as certain other pyogenic organisms. Predisposition is noted in certain conditions, as (1) during or following the occurrence of inflammation, as in lobar and lobular pneumonia. Suppurative infiltration, however, more frequently arises under these circumstances than abscess, and in the rare instances in which the latter occurs it is apt to be comparatively small and multiple. In all forms of inhalation and deglutition broncho-pneumonia, however, abscess of the lung is a frequent sequela.

(2) Perforation of the lung from without or from adjacent organs, as in carcinoma of the esophagus, abscess of the liver, or suppurating hydatid cyst.

(3) Infectious emboli, found in connection with septico-pyemia, frequently cause metastatic abscesses in the lungs. In a mechanical manner they may produce hemorrhagic infarctions, followed by suppuration, or the latter process may occur independently of the former. The abscesses



are usually situated close to the pleura, and are frequently wedge-shaped; they vary in number from one to several hundred, and in size from a pin's head to an orange.

(4) Abscess of the lung may result from inward extension of a purulent pleurisy; and, oppositely, purulent pleurisy may result from an extension of abscess of the lung.

(5) As elsewhere stated (*vide* Pulmonary Tuberculosis), suppuration is quite generally associated with chronic pulmonary tuberculosis.

**Symptoms and Diagnosis.**—The examination of the sputum is of the greatest value in the diagnosis of this disease, since, being purulent, it usually presents a yellow, or less frequently a greenish- or brownish-yellow, color. It emits a feter that is less pronounced than that of either gangrene or putrid bronchitis. Particles of lung-tissue may be visible in the pus, and on microscopic examination of the latter, elastic fibers, the presence of which is of the utmost importance in the diagnosis, may be found in profusion. Next to the investigation of the sputum, the physical signs of cavity are of the greatest assistance in distinguishing abscess of the lung; these, however, are wanting unless the abscess is of a decided size. By themselves, the signs of cavity do not suffice for the recognition of abscess, but when combined with the characteristic sputum leave no room for doubt. The history of individual cases is of considerable importance, as confirming the more characteristic features. Thus antecedent pneumonia or septico-pyemia would be strongly corroborative.

**Prognosis.**—The prognosis is often hopeless, as, for example, when the disease is associated with pyemic processes in other parts of the body. On the other hand, those rare instances in which it is secondary to pneumonia give a comparatively favorable prognosis.

**Treatment.**—The chief aim in the therapeusis should be to support the system by the administration of tonics, stimulants, and antiseptics, as well as by methodical feeding with light and concentrated forms of nourishment. Inhalation of antiseptic sprays (creasote, thymol) should be tried. When the abscess is situated near the periphery of the lung, surgical interference is to be advised as soon as the first indications of increasing weakness appear. For the details of the operation of pneumonotomy for pulmonary abscess the reader is referred to special works on surgery. The statistics of Eichhorst,<sup>1</sup> showing its favorable results, may, however, be mentioned, as follows: in 13 operations recovery or improvement was noted in 6, while fatal terminations occurred in 7.

## PNEUMONOKONIOSIS.

(*Anthracosis, Chalicosis, etc.*)

**Definition.**—A form of chronic interstitial pneumonia that arises from the inhalation of dust-like particles. Different terms have been applied to the condition according to the nature of the dusts inhaled, the chief among these being—(1) Anthracosis (coal-miner's disease),

<sup>1</sup> *Specielle pathologie*, Bd. 1, S. 519.

due to the inhalation of coal-dust; (2) Chalicosis (stone-cutter's phthisis), caused by the inhalation of mineral dusts; and (3) Siderosis, caused by inhaling metallic particles, particularly iron oxid.

(1) **Anthracosis.**—Among dwellers in cities a moderate degree of pigmentation of the lung-tissue with coal-dust is the rule, while in those residing in rural districts the condition is decidedly less common. True anthracosis, however, has reference to such an accumulation of the carbon particles as can be due only to the inhalation of a well-laden atmosphere, or under circumstances when the mucous membrane is unhealthy or without perfect ciliary action. Under such circumstances the normal scavengers of the respiratory organs—the mucous corpuscles lining the trachea, the bronchi, and the alveolar cells—fail to deal successfully with the numerous dust-particles that gain entrance along with the inspired air; hence some of the latter pierce the mucosa and reach the lymph-spaces and lymph-vessels. On reaching the bronchial mucosa they become enclosed in leukocytes, mucous corpuscles, and alveolar cells, and are conveyed by the latter to a more remote destination. Arnold shows that after the particles enter the lymph-system they are carried “(a) to the lymph-nodes surrounding the bronchi and blood-vessels; (b) to the interlobular septa beneath the pleura, where they lodge in and between the tissue-element; and (c) along the larger lymph-channels to the substernal, bronchial, and tracheal glands, in which the stroma-cells in the follicular cord dispose of them permanently and prevent them from entering the general circulation.” Rarely the carbon particles may find their way into the general circulation; this may occur, as shown by Weigert, when the pigmented bronchial glands become adherent to the pulmonary veins, thus giving opportunity for the escape of the carbon granules into the blood.

Anthracosis leads, primarily, to chronic bronchitis, to be soon followed by emphysema; but it must be recollected that extensive anthracosis may be present without any other changes in the lung than the presence of carbon particles stored in the protoplasmic cells. The lung-tissue presents great variations in its degree of susceptibility to these foreign particles. Sooner or later, there is usually produced, as the result of their irritant action,<sup>1</sup> a proliferation of the connective-tissue elements—*i. e.* a chronic interstitial inflammation. This fibroid change usually starts in the peribronchial lymph-structures, though the bronchial and tracheal glands are, as a rule, similarly involved at a comparatively early period. The affected lung-tissue is frequently coal-black, dense, and airless. The pneumonokoniotic areas vary greatly in size and numbers, and not infrequently they coalesce, in which case large portions of the lung-tissue may become the seat of fibroid change. The alveolar walls are observed to be much thickened in some instances, and firm pleuritic adhesions exist. Bronchiectatic cavities may be present, and later necrotic softening of the indurated areas occurs, leading to the formation of small cavities that contain a dark fluid. When the latter communicate with the bronchi their walls are prone to ulcerate. I have noticed that the process almost invariably terminates in pulmonary tuberculosis, and par-

<sup>1</sup> Cohnheim contends that coal particles do not produce irritative changes in the lung, and that the latter are due to irritating substances inhaled with the particles of coal.

ticularly is this true of cases that follow the inhalation of mineral and vegetable dusts.

(2) **Chalicosis.**—Changes similar to those previously described are induced in the pulmonary connective tissue by the inhalation of stone-dust by those who follow certain occupations, such as stone-cutting, knife- and axe-grinding, and millstone-making. The irritating properties of this form of dust cannot be denied, as shown by the great disposition in this subvariety of pneumonokoniosis to the formation of fibrous nodules and diffuse areas of sclerosis in the lungs. The nodules have a gray center and a darker periphery; they are exceedingly dense, and sections are made with much difficulty. The cut surface may present a grayish and distinctly glistening appearance.

(3) **Siderosis.**—This term implies a collection of iron oxid in the lungs, also due to the pursuit of certain occupations (dyeing, iron-smithing, etc.). Cases of much the same nature are caused by the inhalation of vegetable dusts by grain-shovellers, cotton-spinners, cigar-makers, etc. The *pathological* changes are identical with those in anthracosis, though the color-appearance is red instead of black.

**Symptoms.**—Rarely the onset is marked by the symptoms of acute, followed by those of chronic, bronchitis; but in a vast majority of instances chronic bronchitis gradually develops after long exposure to the action of the exciting cause. The symptoms of emphysema are soon superadded, the patient now suffering from dyspnea, and less frequently from asthma. The sputum is diagnostic in anthracosis, being quite dark; in chalicosis a microscopic examination is essential to show the particles of silica; while in siderosis the expectoration presents a reddish color. Apart from the foreign particles, the sputum is for a long period of years muco-purulent in character, and later it often contains the tubercle bacillus.

The physical signs are not distinctive, being identical with those met with in chronic bronchitis associated with emphysema, and followed by those of interstitial pneumonia, and sometimes by those of cavity.

The **diagnosis** is to be made both from the history and from a gross or microscopic examination of the sputum. It may be confirmed by the invariable presence of the signs of bronchitis and emphysema, as well as by the effect of removal to an atmosphere free from dust. In the later stages the detection of infallible evidences of phthisis only serves to corroborate the early diagnosis of pneumonokoniosis.

The **prognosis** is favorable in hygienic surroundings until the more advanced stage is reached. The condition favors the invasion of new growths (lympho-sarcoma, or cobalt-miner's disease; *vide supra*).

**Treatment.**—A change of occupation or several hours of exercise in the open air daily for those who are exposed to dust in work-rooms should be advocated.

The active treatment is the same as for chronic bronchitis and emphysema from other causes, and is to be appropriately modified when pulmonary tuberculosis develops.



## NEW GROWTHS OF THE LUNGS.

## CARCINOMA OF THE LUNG.

ALL varieties of carcinoma have been met with in the lung, but, with rare exceptions, carcinoma of this organ is secondary to similar growths in other parts of the body. To explain its origin it may safely be assumed that the primary new growth involves a vein or lymph-channel, and that the latter carries the germ of the disease to the lung. It is also to be recollected that it may result from extension, or by contiguity from neighboring organs (as the esophagus, mamma, pleura, or mediastinum).

**Etiology.**—The causes of primary carcinoma of the lung must be, in the main, identical with those of carcinoma in general, and are as yet unknown. Most cases occur in middle-aged persons, and, while sex has no influence upon the appearance of the primary form of the disease, the secondary form is more frequent in the female than in the male. In the female secondary carcinoma of the lung is often preceded by carcinoma of the breast. We may also regard hereditary influence as a potent predisposing factor. Secondary carcinoma of the lung is most commonly consecutive to primary carcinoma of the bones, and of the digestive and urinary tracts.

**Pathology.**—The pathological varieties of the primary form are scirrhus, encephaloid, and epithelioma, and of these the latter is the most common. *Primary carcinoma* is usually unilateral, the tumors attaining to a massive size and frequently involving the greater part of one lung. Their favorite seat is in the upper part of the right lung, though the pleura is quite often invaded by the carcinomatous process. Less frequently there is pleurisy with sero-fibrinous exudate, which may be hemorrhagic. Carcinomatous involvement of the cervical, bronchial, and tracheal lymph-glands is quite usual, and rarely even the inguinal glands become implicated. *Secondary carcinomata* are, as a rule, multiple, and may be miliary in size. They are disseminated widely throughout both lungs, though in the rarest instances they may be unilateral. In the softer varieties the central portion of the tumor-mass may undergo fatty degeneration, with subsequent discharge through adjacent bronchi.

**Symptoms.**—The symptoms of carcinoma of the lung vary according to the location and extent of the disease. Among the most marked symptoms belongs pain, particularly when the pleura is implicated. As a rule, for a considerable period of time the symptoms of bronchitis obtain, and later the breathing-space is diminished sufficiently to excite dyspnea and cyanosis. With the increase in size of the new growth compression of the heart, aorta, and large veins may result, whereupon disturbances of the circulation will arise. The new growth may exert pressure on the esophagus, causing dysphagia; or upon the recurrent laryngeal nerve, causing aphonia and hoarseness; or on the trachea or a main bronchus, followed by the symptoms of stenosis of those organs. There are cough and expectoration, the latter frequently containing blood-corpuscles with mucus, and resembling in appearance currant-jelly; the sputa may also rarely exhibit a grass-green color, due to transformation of the blood-pigment. In carcinomatous lungs putrefactive changes sometimes take place, and if so the expectoration and breath

emit an offensive odor, while a microscopic examination of the sputum frequently discloses the presence of carcinomatous elements. The well-known cancerous cachexia invariably develops.

**Physical Signs.**—These will naturally depend upon the extent and location of the new growth. *Inspection.*—If the lung-tissue be extensively involved, the walls of the thorax become unduly prominent and fixed over the seat of the tumor. Indeed, the tumor may, though rarely, protrude between the ribs. The intercostal spaces are widened, and the superficial veins, in view of the fact that they cannot empty themselves into the internal veins, appear engorged; from the same cause edema affecting the thorax, neck, face, and arms may be noted. Swelling of the lymph-glands in the neck or axilla is often witnessed, and is a symptom of high importance. On *palpation* the tactile fremitus may be found to be diminished or absent. The *percussion-note* will be flat, since the air-vesicles and smaller bronchi are replaced by the solid growth. On *auscultation* friction-sounds are the rule. The respiratory sounds may be greatly enfeebled or absent; but if the carcinomatous tumor communicates with a wide-mouthed bronchus, bronchial breathing may be audible, and the usual physical signs of lung-cavity may be developed. The signs of general bronchitis are present in most instances, being most pronounced in the secondary or disseminated form of the disease; in the latter variety the lung may shrink, forming a condition in which retraction of the chest-walls on the affected side must ensue. If pleurisy with effusion occurs as a secondary event, the detection of the characteristic cancer-cells in the contents of the pleural cavity will show the precise nature of the thoracic affection.

**Diagnosis.**—The following symptom-group will pretty well establish a diagnosis: A peculiarly shaped dull area (as when it extends under the sternum), perhaps a marked prominence over the site of the tumor, enlarged and hard lymphatic glands in the vicinage, and more or less of the compression-symptoms—circulatory, nervous, bronchial, or trachial stenosis. In rarer instances the diagnosis may be made by the occurrence of metastasis to the chest-wall. Again, the discovery of cancer-tissue in masses accidentally detached gives reliable indication of the disease.

The **differential diagnosis** between pulmonary carcinoma and *pulmonary tuberculosis* can be made with positiveness only by a careful microscopic examination of the sputum. From *fibroid induration* of the lung it is easily discriminated, owing to the history and slower course of the latter affection.

**Prognosis.**—This is bad, as death may occur suddenly from abundant hemorrhage or more frequently from either exhaustion or asphyxia. The duration of the affection varies from six months to a year, or, rarely, even two years.

**Treatment.**—The treatment must be addressed solely to the relief of pain and other subjective symptoms.

#### SARCOMA OF THE LUNG.

Primary sarcoma of the lung is rare, but in instances of generalized sarcomatosis the lungs show larger or smaller nodules “in almost every

case" (Birch-Hirschfeld), occurring in connection with osteo-sarcoma of other organs or in lympho-sarcoma of the cervical glands.

Secondary sarcoma, occurring in consequence of invasion of the root of the lung by sarcomatous disease of the post-bronchial glands, is also a not uncommon condition.

Neoplasms occurring among the cobalt-miners of Schneeberg were described by Hesse and Tragner as lympho-sarcomata—slowly growing masses that attained to a large size and gave metastasis to lymph-glands, pleura, liver, and spleen. In a majority of these cases there was an associated pneumokoniosis, which had probably predisposed to the new growth.

#### HYDATID CYST OF THE LUNG.

Hydatids in the lungs may either be primary or secondary, the former variety being exceedingly rare, and the latter somewhat less so. Almost invariably the echinococci are developed in other organs—the liver in particular—and find their way to the lungs, either by direct perforation through the diaphragm or by entering through the blood-current.

The **etiology** and **pathology** will be considered at sufficient length in connection with Hydatid Cysts of the Liver.

**Symptoms.**—The clinical manifestations are quite varied, even though the cyst may entirely conceal itself. It is important to recollect that similar involvement of the liver usually coexists; and in addition to the symptoms of the latter affection there may be pain in the chest, dyspnea, considerable cough, and, rarely, blood-stained expectoration.

The **physical signs**, when present, are as follows: Diminished vocal fremitus, defective expansion, dulness on percussion with an absence of the respiratory murmur, and later signs of cavity-formation may appear.

A positive **diagnosis** of hydatid cyst of the lung can be made only when the scolices, pieces of membrane, and the hooklets of the echinococcus are demonstrable in the sputum. Besides being evacuated into the bronchi, the cysts may rupture into the adjacent serous sacs (pleura, peritoneum, pericardium), or externally, the latter being the most favorable mode of termination. Unless they are discharged early by ulceration into the bronchi or externally, they are apt to excite inflammation of the adjacent lung-tissue and tubes, accompanied by an active febrile movement and an aggravation of the symptoms before detailed: these complicating conditions may assume a dangerous form, or the patient may, if the growth attains large dimensions, become asphyxiated.

**Prognosis.**—The affection is always attended with great danger, and is of more serious import when secondary to involvement of the liver than when primary.

**Treatment.**—When it can be shown that the growths are situated at the periphery of the lung operation should be carefully considered. The physician stands powerless to do more than to relieve urgent symptoms in special cases and to support the vital functions.



## V. DISEASES OF THE PLEURA.

## PLEURISY.

*(Pleuritis.)*

**Definition.**—An inflammation, either local or general, of one or both pleural membranes. The disease, as shown by postmortem examinations, is of great frequency.

**Varieties.**—Pleurisy has been variously classified. *Etiologically*, the distinction between primary and secondary forms of the disease should be made, as well as a division into tuberculous, carcinomatous, septic, etc. *Pathologically*, all cases may be summarized under the following heads: Localized and generalized and dry (plastic) pleurisy and pleurisy with effusion (sero-fibrinous, purulent, hemorrhagic). They may also be classified according to their duration into acute, subacute, and chronic pleurisies. I shall describe the following forms, which are based partly upon their etiologic and clinical course, though mainly upon their pathologic manifestations—viz. (a) acute plastic pleurisy; (b) sero-fibrinous pleurisy; (c) purulent pleurisy (empyema); and (d) chronic adhesive pleurisy.

**Bacteriology.**—In all forms of the disease the immediate causes are various micro-organisms or their irritating chemical products. Conspicuous among these is the bacillus of tuberculosis, even, though rarely, found in the pleuritic exudate. Inoculation of guinea-pigs with the latter by Eichhorst gave positive results in 15 out of 23 cases that were considered to be of the primary form. Although rarely containing bacteria, Netter, Prudden, and others have found in the exudation of fibrino-serous pleurisy the streptococcus pyogenes, the staphylococcus, the typhoid bacillus, and the diplococcus of pneumonia. The micro-organisms most commonly present in emphysema are the micrococcus lanceolatus and the streptococcus, the former especially in the pleurisy associated with pneumonia (in two-thirds of the cases occurring in children—Levy), and the latter in those independent of pneumonia, particularly in adults. Among other bacteria that have been found rarely in the effusion are the colon bacillus, the proteus vulgaris, the gonococcus, Friedländer's bacillus, and various saprophytic bacteria. Except in the case of the pleuritic exudation (usually purulent) in pneumonia, in which the diplococcus is alone present in about one-half of the cases, the afore-mentioned micro-organisms are generally found in association.

## ACUTE PLASTIC PLEURISY.

*(Dry, Fibrinous Pleurisy.)*

**Pathology.**—The lesions are usually circumscribed, the part inflamed being intensely injected. It has lost its natural lustre, and instead has a dull, non-glistening surface "like a tarnished mirror," due to a slight fibrinous exudate. Minute ecchymoses are seen at different points. Later the exudate may become more copious, when the

pleura presents a rough, shaggy appearance. On account of the friction between the two pleural membranes in high grades of dry plastic pleurisy, the exudate may be very thick, and its color-appearance is then yellowish- or reddish-gray. This sheeting of fibrinous exudate entangles in its meshes numerous embryonic round cells, out of which blood-vessels and connective tissue are developed. The opposing surfaces of the pleura adhere. Occasionally, in the lighter grades, the disease does not advance to firm adhesion, and in such instances the products of the exudate undergo fatty degeneration and are absorbed. The respiratory movements are but little disturbed in these cases.

**Etiology.**—The affection may be (a) primary or (b) secondary. (a) By the *primary form* is meant an inflammation of the pleura occurring in previously healthy persons. It is exceedingly rare, and doubtless many instances of true secondary pleurisy are regarded as belonging to this category, inasmuch as pleurisy may exist for an indefinite period without exciting noticeable symptoms. Of great etiologic prominence is exposure to cold and wet, and next to this stands mechanical injury. It is more common in men than in women, and especially during the time of active life, on account of the greater degree of exposure of the former than the latter sex. In almost all instances a careful search will disclose the existence of some diathesis (tuberculous, gouty, rheumatic) that may be properly regarded as the favoring cause. The changeable weather of the winter and spring augments the proportion of cases during these seasons as compared with summer and autumn.

(b) The *secondary form* of dry plastic pleurisy arises from extension of acute and chronic inflammatory affections of the lungs and other neighboring organs. Hence it frequently follows croupous pneumonia, somewhat less frequently broncho-pneumonia, and more rarely still hemorrhagic infarct, abscesses, and pulmonary carcinoma and gangrene. When pleurisy occurs on the right side it must be recollected that it may have originated in inflammation of the liver. Plastic pleurisy sometimes arises in acute articular rheumatism, to which it may essentially belong. It is an almost constant secondary process in chronic pulmonary tuberculosis, and may, though rarely, even constitute the primary lesion (primary tuberculous pleurisy). The disease may appear as a complication in chronic alcoholism and in chronic Bright's disease. Finally, inflammation of other serous membranes, as of the peritoneum and pericardium, by direct extension through the lymphatics of the diaphragm, invade the pleura.

**Symptoms**—The affection may vary in intensity between the extremes of mildness and great severity, though, as a rule, well-marked local symptoms attend the onset. Among the latter a sharp "stitch" in the side, that is usually referred to the nipple, is the most prominent. The pleural pain is increased by inspiration as well as by voluntary motion of the affected side, and hence the patient assumes a fixed position in which he favors the affected side by leaning toward it. There is a dry, distressing cough that is restrained for obvious reasons, and the respiration is somewhat hurried, painful, and jerking in character until the exudation is poured out, when relief from this and other local symptoms ensues.

The general symptoms are not pronounced, and, save in comparatively rare instances, do not correspond with the local signs. The temperature is not typical, rarely exceeding  $103^{\circ}$  F. ( $39.4^{\circ}$  C.), and more often it is below  $101^{\circ}$  F. ( $38.3^{\circ}$  C.). The pulse is usually small and tense or soft in character, registering from 90 to 120 beats per minute. Not infrequently the cases are so mild as to be attended by few, if any, subjective symptoms. The patient may complain of ill-defined, uneasy sensations in the affected side, but does not discontinue his usual occupation. On the other hand, the worst cases of acute plastic pleurisy—which, fortunately, are rare—manifest violent symptoms: there is a distinct chill, a speedy development of high fever ( $104^{\circ}$  F.— $4.0^{\circ}$  C.), and profound prostration, and the general and local symptoms are proportionately aggravated. The illness then is often a fatal one.

**Physical Signs.**—On *inspection* the movements of the chest-wall on the affected side are observed to be much restricted, particularly during the first day of the affection. During a later period *palpation* confirms the results of inspection, while *percussion* yields a normal note. *Auscultation* renders audible a grazing friction-sound, which, though audible, is most intense at the end of inspiration.

With the occurrence of fibrinous exudation *palpation* detects over the corresponding area a diminution of the tactile fremitus. On *percussion* there is, as a rule, a slight though variable degree of dulness; and on *auscultation* the crepitating or rubbing friction-sounds are heard both on inspiration and expiration, being intensified by deep breathing. These sounds frequently endure for a day or two after the other symptoms have disappeared. Very rarely the plastic exudation may be so extensive as to cause compression of the lung, in which instance the breath-sounds may become bronchial in character; and I have known a case of this sort to be mistaken for lobar pneumonia. In addition, the breath-sounds will be feeble and distant.

**Diagnosis.**—By exercising ordinary care the clinician can scarcely mistake other thoracic affections for dry pleurisy, the latter being diagnosed to a certainty by the presence of the characteristic friction-murmur. *Intercostal neuralgia* may present features not unlike those of acute pleurisy. In both affections there is frequently a history of exposure, followed by severe chest-pains that are excited by coughing and deep breathing. In neuralgia, however, there are painful pressure-points, and the pleuritic friction-sound does not occur. *Pleurodynia* may also give a history very similar to that of acute pleurisy, but the presence of the characteristic physical signs of pleurisy are absent.

**Prognosis.**—The duration of the affection varies from a few days to three weeks, and the immediate outcome is favorable as a rule. It cannot be doubted, however, that a primary attack predisposes to subsequent attacks, and thus, as a result of repeated seizures, pleural thickening and intrapleural adhesions often arise. Lung-expansion may in this manner be restricted, with the gradual development of interstitial pneumonia as a consequence. Acute plastic pleurisy is not infrequently a terminal condition in serious forms of illness (*e. g.* septicopyemia and chronic nephritis).

**Treatment.**—The first object in the treatment is to relieve the pain, and this can best be accomplished by the hypodermic use of mor-



phin. The inflammatory process is best controlled by absolute rest in the recumbent posture, allowing the patient to assume that position which gives him most comfort. I am also in the habit of administering moderate-sized doses of quinin (gr. iv—0.259—three times daily). After the exudation has appeared, the iodids of iron and potassium, in combination, may be employed. Locally, nothing is so effective as cold in the form of the ice-water bag or Leiter's coil, preceded, in robust patients, by the local abstraction of blood (3iij to vj—96.0–192.0) by leeches. At the end of one week the morphin may usually be discontinued. During convalescence the patient should be instructed to take deep inspirations several times in succession, not less than a dozen times each day, with a view to obviating so far as possible the pleural adhesions and other unfavorable consequences. Symptomatic anemia may be present at this time, and should be met by iron given internally. At this time iodine may be used locally with great benefit; I have not, however, seen any favorable results from blisters. For the pain which continues in the side after all detectable physical signs have disappeared the use of the constant current over the seat of the pleurisy for twenty minutes at a time gives almost instantaneous relief (Loomis).

#### SERO-FIBRINOUS PLEURISY (PLEURISY WITH EFFUSION, SUBACUTE PLEURISY).

**Pathology.**—During the first stage of sero-fibrinous pleurisy the changes are the same in character as those met with in dry pleurisy, though of severer grade, and usually involving the greater portion of the pleura on the side affected. There is an abundant exudation of serum, and usually the entire pleura becomes coated with a fibrinous exudate, that varies greatly in thickness and arrangement. The latter is thin and smooth in some instances, though more frequently it forms a thick layer, presenting a shaggy surface on the one hand or an irregular, honeycombed surface on the other. Lymph in the form of flocculi is rather abundant in the serous effusion. The interlobular pleural surfaces are also invaded as a rule, in consequence of which they become adherent. The fluid exudate varies greatly in quantity ( $\frac{1}{2}$  to 8 pints—4 liters), is often of a citron color, and is, in the majority of instances, clear or slightly turbid. Rarely it is of a dark-brown color.

Unless adhesions between the pleural surfaces have previously existed the effusion gravitates to the most dependent portion of the pleural cavity. Microscopically, there are found leukocytes, red blood-corpuscles, endothelial cells, threads of fibrin, and, rarely, crystals of cholesterin and uric acid. The composition of the fluid is almost identical with that of blood-serum, and on boiling it is found to be rich in albumin. Spontaneous coagulation may take place on standing.

*Changes in the Neighboring Organs.*—So long as the normal retraction of the lung is not overcome by the fluid that collects in the pleural cavity, the latter does not produce positive intrathoracic pressure, and hence does not produce displacement of adjacent organs. It may be assumed that until the pleural sac is at least one-half filled with sero-

fibrinous exudate the natural contractility of the lung is not destroyed. At this period there may be a slight displacement of the mediastinum toward the opposite side, due to traction exerted by the normal retractility of the sound lung. Obviously, large effusions must in a mechanical manner displace the pleural membranes, thus causing compression of the pulmonary structures lying above the effusion. A very copious effusion may push the lung up and back against the vertebral column and convert it into a small, flat, bloodless, and airless mass (atelectasis). While a total absence of air in the collapsed lung is due chiefly to compression by the fluid, to some extent, however, the air may be absorbed by the vessels or even by the effusion (Strümpell).

Together with compression of the lung by the effusion, pressure is also exerted by the latter against the mediastinum, causing displacement of the heart. The mediastinum also loses the normal traction-force of the lung upon the affected side, and hence the lung on the sound side draws the mediastinum toward itself by its own retractile energy. Osler shows that even in the most extensive left-sided effusion the heart's apex is not rotated, but that the normal relative position of the apex and base obtain, though the apex is in some instances lifted, and in others the heart lies more transversely. The right chambers of the heart occupy most of the interior of the organ, showing that the displacement of the mediastinum with the pericardium and its contents to the right involves no appreciable twisting of the heart itself.

Downward displacement of the diaphragm takes place in extensive effusion, and shows itself on the right side by the lowering of the liver to a variable distance below the inferior costal border; on the left side large effusions produce pressure-displacement of the stomach and the transverse colon, and, to a slighter extent, of the spleen. It must be recollected that adhesions may prevent displacement of any of the adjacent organs.

**Etiology.**—In the present state of our knowledge the causal factors are identical in nature with those producing dry plastic pleurisy, the pathologic differences being attributable to the differences in the intensity of the processes. It is highly probable that the degree of severity is dependent upon the previous condition of the patient, whether he be suffering from some other affection or not, and upon the amount of specific poison gaining access to the pleura.

The affection may be primary, but is much more often secondary; and this fact may be explained by reference to any of the specific micro-organisms producing the affection. Many of the cases follow quickly upon exposure to cold or wet or an injury to the thorax. I thoroughly agree with those authors who contend that about three-fourths of the cases of sero-fibrinous pleurisy are induced by tuberculous infection of the pleura. The tuberculous process may invade the pleura primarily, but more often it is secondary to tuberculosis of the lungs; less frequently, though more often than is generally supposed, it is secondary to tuberculous peritonitis. In these instances the tubercle bacilli probably find their way from the peritoneum to the pleura by traversing the lymphatics in the diaphragm. I am convinced that a large percentage of apparently primary cases of tuberculous pleurisy have their origin in a circumscribed and more or less



latent tuberculous focus in the lungs. It is not improbable also that tuberculous processes in other viscera may furnish the tubercle bacilli for secondary pleural infection. Moreover, the fact that many cases of sero-fibrinous pleurisy recover does not disprove their tuberculous nature. The typhoid bacillus of Eberth has also been known to provoke pleurisy (Bozzolo, Fernet, and others).<sup>1</sup>

The affection is not infrequently secondary to acute articular rheumatism, which is itself most probably a microbic affection. It also arises as a complicating condition in the course of various acute and chronic affections of the chest, as pericarditis and catarrhal pneumonia, and may develop in acute infectious diseases, as typhoid fever or lobar pneumonia. It may occur as a complication in the chronic affections of various viscera (chronic nephritis, cirrhosis and carcinoma of the liver). The predisposing causes are the same as for the dry plastic form.

**Symptoms.**—The description here refers particularly to primary sero-fibrinous pleurisy, and it is important to recollect that when secondary to other acute and chronic affections characterized by great bodily weakness the pleuritic symptoms may be more or less completely veiled.

With few exceptions the onset is insidious, the symptoms being quite mild, but rarely there is a sudden onset with active symptoms (rigor, high fever). In the majority of instances the patient first complains of a stitch-like pain in the side; this is rarely pronounced, but is aggravated upon deep breathing and upon any muscular exertion. Dyspnea soon arises and gradually increases in intensity. Cough may be present or absent, and in some instances is attended by a scanty mucoid expectoration that may rarely be blood-streaked.

The constitutional symptoms are of correspondingly slow and gradual development. From the commencement of the attack a moderate febrile movement at night may be observed, and the pulse will be found to be frequent, small, and compressible, or, more rarely, tense. At the time of the patient's first visit to his physician he may give a history of having gradually lost flesh and strength for a period of weeks together, though he may not have been obliged to abandon his vocation. He looks pale, his countenance wears an anxious expression, and he is without appetite. These cases frequently drag along from two to four weeks before consulting a physician, the local symptoms going unnoticed, and the patient making complaint only of weakness, anorexia, headache, etc.

Sometimes the more acute symptoms characterize the period of invasion, and, after lasting a few days, exhibit a decided remission; but at another subsequent period there may be a sudden recurrence of the local and general phenomena, and particularly of the dyspnea. The pleural cavity, which may have been one-half or two-thirds full, now becomes completely filled.

**Special Symptoms.**—*Pain.*—Chest-pain is an almost constant but not highly characteristic symptom, and, though usually among the earliest symptoms, it may not be present until a few hours or a day after the commencement of the affection. It may be described as a sharp, shooting pain, and is popularly termed a "stitch in the side." It may, however, be tearing or dragging in character. Its intensity is not a safe in-

<sup>1</sup> *Annual of the Universal Medical Sciences*, 1892, vol. ii. p. 12.



dication of the severity of the disease. It is usually referred to a small spot below the nipple or to the mid-axillary region; exceptionally, however, it is more diffuse, and in my experience it has not infrequently been retrosternal or referred to limited areas below the inferior costal border. When absent it may be excited by coughing, sneezing, deep inspiration, and stooping. With the appearance of the effusion the pain diminishes, and, as a rule, soon disappears.

*Dyspnea.*—The respiration is shallow, catching in character, and hurried in consequence of the severe pleural pain; in copious effusions, that render one lung almost or wholly functionless, the dyspnea may become intense, even attaining to well-marked orthopnea. It reaches its most pronounced form in persons who have previously been robust, and in those in whom the effusion has developed rapidly. On the other hand, when the pleural sac fills slowly dyspnea may be absent, except on exertion. This symptom appears frequently before the effusion takes place, and is then due partly to the fever and partly to the pleuritic pain. Following marked disturbances in the respiration, cyanosis appears and may become quite pronounced.

*Cough and Expectorations.*—Little need be added to what has already been stated. When there is present much expectoration it is most frequently due to associated bronchitis or to pulmonary tuberculosis; there may, however, be a total absence of expectoration, and in such instances the exciting cause of the cough is probably the pleuritis. Both the cough and expectoration are apt to be increased during the process of resorption of the exudate as the result of a catarrhal bronchitis that is apt to develop in the re-expanding lung.

*Fever.*—The rise of temperature is not rapid as a rule, nor does it reach a high point ( $101.5^{\circ}$  to  $103^{\circ}$  F.— $38.6^{\circ}$ – $39.4^{\circ}$  C.). At the end of a variable period—usually one to three weeks—the temperature falls by lysis, and soon touches the normal. The temperature may be of the continued type in many acute cases. In subacute forms the temperature may rarely rise above  $101^{\circ}$  F. ( $38.3^{\circ}$  C.), or may, finally, assume a hectic type. The surface-temperature of the affected side is from one-half to two degrees ( $.4^{\circ}$ – $1.6^{\circ}$  C.) higher than that of the normal side.

*Pulse.*—The pulse is quickened, beating 100 or more per minute, and its volume and tension are diminished. Irregularity both of the volume and rhythm of the pulse may also be observed. These pulse-characteristics are to be attributed to the pressure of the effusion upon the heart and great vessels.

*Gastro-intestinal Symptoms.*—Loss of appetite is commonly present, and more rarely nausea and occasional vomiting may be met with at the outset. Constipation is the rule.

*Renal Symptoms.*—The amount of urine is diminished both during exudation and while the exudate remains at the same level. The daily quantity may not exceed eight or ten ounces, but the specific gravity is increased, ranging from 1018 to 1028. An increase in the daily amount of urine excreted is frequently the first sign of commencing absorption of the exudate, and the rapid resorption of the copious effusion may greatly augment the flow of urine to 80 or 100 ounces (2.5 to 3 liters) daily (Strümpell). The cause of the diminished secretion of urine is, in the main, diminished arterial pressure.

**Physical Signs.**—The physical signs of sero-fibrinous pleurisy differ with the amount of effusion present, and also with the particular stage of the affection: those of the first stage, however, are identical with the signs pointed out in connection with dry plastic pleurisy, and need not be restated here. We will note the physical signs (1) during the stage of effusion, as well as (2) those presented when resorption of the effusion has taken place.

(1) *Stage of Effusion.*—When the pleural sac is only partly filled there is noted, on *inspection*, but little change in the thoracic contour. The respiratory movements are, however, restricted, owing to mechanical hindrance to the lung-expansion. In the majority of instances the effusion increases until positive intrathoracic pressure and noticeable bulging in the middle and lower third of the chest-wall on the affected side take place; the intercostal spaces below are widened and more or less nearly effaced. The apex-beat of the heart is displaced, being visible in right-sided pleurisy to the left of the vertical mammary line in the fourth and fifth interspaces, and in left-sided pleurisy to the right of the right mammary line, or even beyond, in the third and fourth interspaces. The apex of the heart may take a position behind the sternum, when no impulse will be visible.

*Palpation.*—The limited movement of the chest is readily appreciated on palpation, and in large effusions the chest-wall is practically fixed. The separation of the ribs and the obliteration of the intercostal spaces are easily made out in the same manner. Edema of the chest-wall is rarely present, and fluctuation almost never. An important and early physical sign is the diminished tactile fremitus, which is soon abolished, except in infants, in whom it may be excited on crying. This is a less valuable sign in women than in men, owing to the differences in the vocal vibrations in the two sexes. In copious effusions tactile fremitus may sometimes be obtained when bands of adhesion, which serve as a medium for the transmission of local fremitus, connect the pulmonary with the costal pleura. The apical impulse can also be readily located by palpation. The displaced spleen or liver can be readily felt through the abdominal wall, and must not be mistaken for an actual enlargement of these organs.

*Mensuration.*—It must not be forgotten that in right-handed adults the right side is, normally, slightly larger than the left; and it is only after the effusion is considerable in amount that the cyrtometer shows any alteration in the thoracic contour or an enlargement of the affected side. The tape, however, exhibits the difference in expansive motion of the two sides early, or when there is a moderate amount of fluid. At the end of expiration the circumference of the affected side will be found to be one or two inches greater than that of the left side, while at the end of inspiration the difference will be but slight. The cyrtometric tracing also shows a discrepancy between the horizontal outlines of the two sides.

*Percussion.*—At first the percussion-note is impaired, either posteriorly or in the infra-axillary region, and a little later there is dulness, tending toward flatness (deadness), with increasing effusion. The resistance to the pleximeter-finger becomes greatly augmented. In cases in which the effusion rises to the fourth rib anteriorly there is dulness over



the fluid above and absolute flatness below. Since both the flatness and dulness are due to the fluid, it is obvious that the upper level of the latter must, whenever free, change with the posture of the patient; hence the limit of dulness will be higher in the sitting than in the recumbent position. When the pleural sac is filled or when the effusion is confined by adhesions, the latter sign is not obtainable. If the upper level of the fluid reaches the lower border of the third rib, the percussion-note above the line of dulness is tympanitic or vesiculo-tympanitic (*Skoda's resonance*). This note is most readily elicited in front, though it may also be present behind and above the level of effusion. In copious exudations the cracked-pot sound may be elicited immediately below the clavicle in the usual manner, and "*Williams's tracheal tone*" may sometimes be obtained in large exudations. This may also be obtained near to the spine on the affected side or at a point corresponding to the seat of the compressed lung. When the patient is sitting or in the erect posture, the upper limit of dulness in large effusions is not a horizontal line, but is highest at the spine and falls as we proceed to the front, which is its lowest point. On the other hand, the upper line of dulness in moderate effusions begins "relatively low down in the back, passes upward from the vertebral column, and soon turns upward and proceeds obliquely across the back to the axillary region, where it reaches its highest point; thence it advances in a straight line, but with a slight descent, to the sternum" (Ellis). This curved line resembles the italic letter *S* (Garland). On the right side the flatness is continuous with that of the displaced liver; on the left it passes into and may obliterate Traube's semilunar space.

*Auscultation*—The signs of the first stage have already been described (*vide* Plastic Pleurisy). With the appearance of the effusion the breath-sounds become weak, distant, and have a bronchial quality. Soon the respiratory sounds over the affected side will be entirely absent, except near the upper level of the fluid posteriorly, where distant bronchial breathing and, less frequently, diffuse vocal resonance are audible. The latter sounds may exhibit a metallic or amphoric quality, and may be accompanied by râles (pseudo-cavernous signs). The latter are more frequently met with in children than in adults, and often give rise to a false diagnosis. Above the level of the fluid there is bronchovesicular breathing, and on the opposite side intensified breath-sounds may usually be noted. The effusion-sounds may manifest a nasal or metallic quality, simulating somewhat the bleating of a goat (*Laennec's egophony*). This is best obtained near the upper level of the fluid in large effusions, and at or above the angle of the scapula when the effusion is moderate.

(2) *Stage of Resorption*.—With resorption of the fluid there is a decrease in the size of the affected side, together with a return of the normal appearance of the intercostal spaces and the respiratory movements. In many instances there is positive retraction, leading to thoracic deformity with displacement of neighboring organs toward the affected side; and this retraction may be either general or circumscribed. The inferior intercostal spaces are more or less narrowed; the shoulders droop; the nipple approaches the median line; the spine may be curved, the convexity being directed toward the sound side (quite rarely toward the



affected side); and the scapula projects from the chest-wall on the affected side. In children, and even in adults, the lungs and thorax gradually expand in order to overcome this chronic deformity. On the other hand, the extensive adhesions between the pleural membranes produce permanent shrinkage of the thorax and displacement of respiration.

*Palpation*.—The tactile fremitus closely follows the fluid as it subsides from above downward without any extreme degree of thickening of the pleural membranes, though cohesion of their surfaces may prevent its return over the lower segment. The inspiratory movement of the chest-wall gradually returns, but not to its former limit.

*Mensuration* shows a steady diminution in the size of the side involved, which finally becomes smaller than its fellow.

*Percussion*.—The dull or flat note gives way to normal percussion-resonance, proceeding from above downward in a gradual manner; but the latter is not renewed over the lower portion of the pleural cavity for a long period after the exudation has disappeared. The abnormal areas of flatness due to displacement of organs (liver, spleen, heart) also disappear.

*Auscultation* discloses most important signs during the stage of resorption. The breath-signs reappear at first above, and then lower down, until the base is reached. With commencing subsidence of the fluid the respiratory sounds are feeble and distant, but later they resume their natural distinctness; and partly as a result of the revival of the natural muscular tonicity, and partly in consequence of the disappearance of the fluid, the two roughened pleural surfaces come in contact and play upon one another, giving rise to a rubbing, creaking friction-sound on auscultation. These friction-murmurs may persist for months after the effusion has been absorbed. Occasionally the lower portion of the compressed lung remains permanently inexpandible, and usually in such circumstances the upper portion of the lung is the seat of compensatory emphysema, which is recognizable by the customary physical signs. By auscultation we may note the return of the heart-sounds to their normal position.

### **Special Clinical Forms of Acute Sero-fibrinous Pleurisy.**—

The separate varieties are dependent upon the nature of the effusion and the character of the etiologic factors, and in this connection the main clinical features of a few special types may be briefly described. They are as follows:

(1) **Tuberculous Pleurisy**.—This is, in the majority of instances, secondary to pulmonary tuberculosis. On the other hand, the primary lesions may be situated in the pleural sac and give rise to (1) *Acute sero-fibrinous pleurisy* (with the usual course); (2) *Subacute pleurisy* (with insidious course), leading to tuberculous invasion of the lungs; and (3) *Chronic adhesive pleurisy*, in which the course and physical signs correspond with those that will be depicted in a special section on Chronic Pleurisy.

The morbid lesions are similar to those met with in other forms, plus the specific tubercles, which may be exceedingly numerous (miliary tubercles) on the one hand, or confined to a few circumscribed areas on the other. This variety of pleurisy has no special etiologic connection with empyema, and the effusion is usually sero-fibrinous and often blood-stained.

Brief reference should be made to those instances in which tubercu-

lous pleurisy is followed by tuberculous pericarditis or peritonitis, or both. The two latter affections will be considered elsewhere. Suffice it to state here that tuberculosis of the serous membranes usually pursues a chronic course, lasting a year or more, and exhibits widely varying degrees of intensity in its symptoms in different cases, and from time to time in the same sufferer. We must grant that tuberculous pleurisy may pursue a favorable course with apparent recovery, though too often, after a variable interval of time, tuberculous symptoms are manifested.

(2) **Diaphragmatic Pleurisy.**—This term is applied to those instances in which the diaphragmatic portion of the pleura is involved, either alone or in part. There occurs an exudate that may be either plastic or sero-fibrinous, though rarely large in amount. The *symptoms* are acute, and the pain, which is lancinating in character and situated in the epigastric region, is the most prominent feature. Geuneau de Mussy<sup>1</sup> holds that pain along the tenth rib, extending from the anterior extremity to the sternum and xiphoid cartilage, is pathognomonic. It is increased by deep inspiration and by pressure over the insertion of the diaphragm at the tenth rib, and often abates when effusion takes place. Dyspnea is a marked symptom in most cases, and the patient may be forced to assume a stooping or sitting posture, the respirations being superficial, purely thoracic, and “catching.” Cough, nausea, and even vomiting, may occur. In a case under my own care vomiting, due most probably to associated peritonitis, was a troublesome symptom.

The constitutional features are quite pronounced, and particularly the fever, which exceeds that met with in other forms of pleurisy. The patient's anxiety is extreme. The effusion may be purulent, and if so bulging of the lower intercostal spaces, followed by edema, may occur.

The physical signs are for the most part negative.

(3) **Encysted Pleurisy.**—This term has reference to effusions that are circumscribed in consequence of adhesions between the pleural membranes. There may be two or more pouches, with or without communication. This so-called encapsulated pleurisy may occupy any part of the chest, and is exceedingly variable in extent. The symptoms and physical signs are rarely trustworthy for diagnosis, but should usually afford ground for suspicion, and hence should lead in every instance to the employment of the exploratory puncture.

(4) **Interlobar Pleurisy.**—This variety is usually secondary to, or associated with, the ordinary type of acute sero-fibrinous pleurisy. The serous surfaces, dipping between the lobes, are involved in the inflammatory process, and the fluid becomes encapsulated in this position in consequence of interlobular pleural adhesions. It is more frequent on the right than on the left side, and its favorite seat is near the root of the lung, between the upper and middle lobes. Osler<sup>2</sup> met with a case following pneumonia in which there was between the lower and upper and middle lobes of the right side an enormous purulent collection that looked at first like a large abscess of the lung. Fistulous connection with a bronchus often occurs, and the purulent expectoration that follows may be the first symptom to attract the attention to the process of supuration in the thorax. Prior to the occurrence of this accident the patient gives evidence of indisposition without definite symptoms. The

<sup>1</sup> *Arch. gén. de Méd.*, 1853, vol. xi., quoted by Fox.      <sup>2</sup> *Practice of Medicine*, p. 567.



patient may or may not give a clear history of antecedent pleurisy. These cysts contain, as a rule, but a small amount of fluid, and do not cause much bulging of the intercostal spaces. Indeed, in a case of my own at the Philadelphia Hospital there was actual retraction, though the aspirating needle showed the presence of effusion.<sup>1</sup>

(5) **Hemorrhagic Pleurisy.**—By this term is meant an admixture of

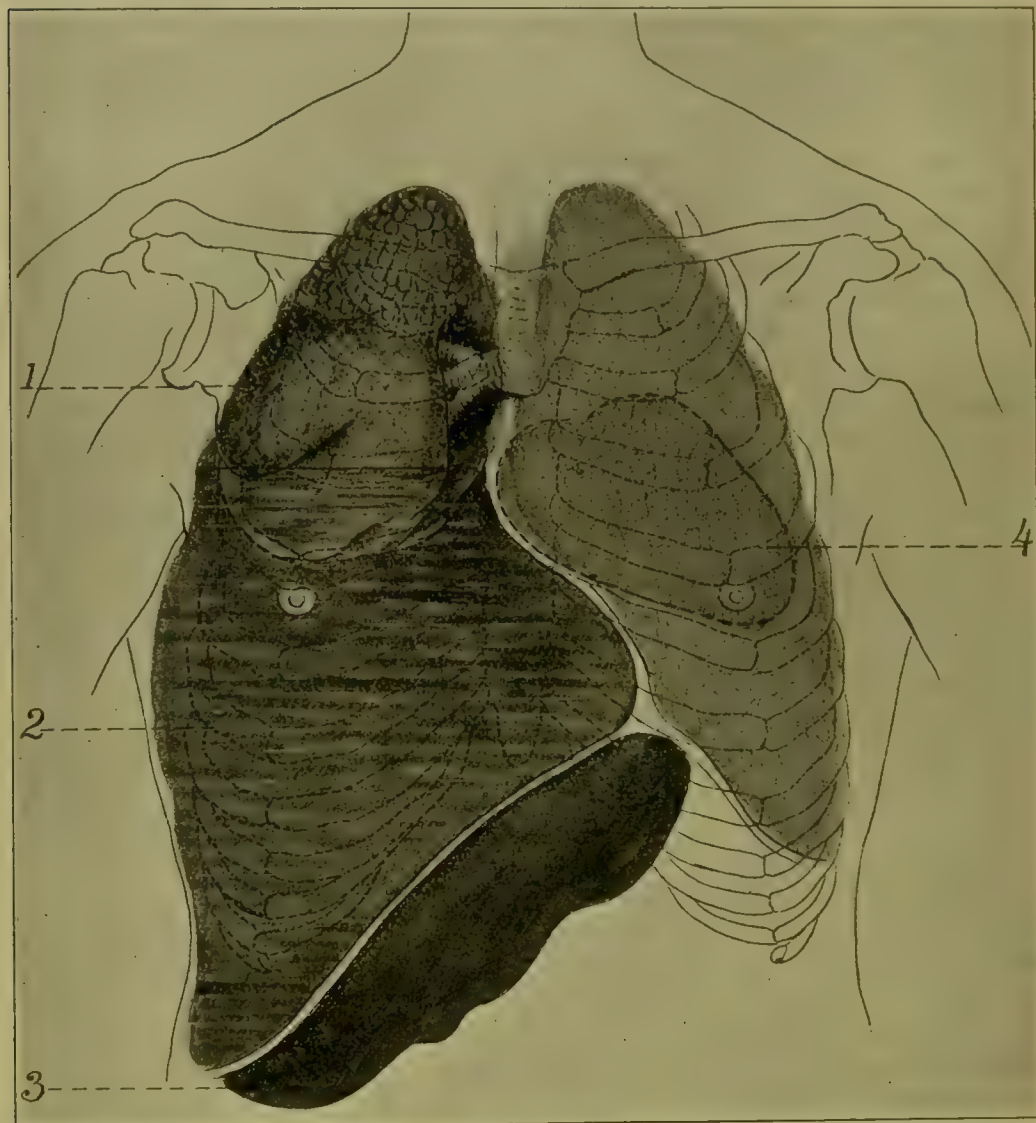


FIG. 47.—Illustrating pleurisy with effusion: 1, compressed lung-tissue, giving dull tympany on percussion; 2, fluid exudation obliterating intercostal spaces; 3, depressed liver; 4, displaced heart.

blood with the exudate in acute sero-fibrinous pleurisy, in quantities sufficient to be detectable by the unaided eye. The condition must be separated from hemothorax. The causes of hemorrhagic pleurisy are—(1) Tuberculous infection, either of the miliary or the chronic (circumscribed) form, following tuberculous disease of the lung; (2) Carcinoma of the pleura; (3) Bright's disease, and cirrhosis of the liver; (4) Adynamic states of the system associated with malignant forms of acute infectious diseases.

<sup>1</sup> *International Clinics* (1894), vol. i. p. 39.



In a certain proportion of the cases no assignable cause can be found, and if the condition be observed for the first time after aspiration, the fact that it may have been engendered by an accidental wound of the lung must be remembered.

**Diagnosis.**—In diagnosing pleurisy our attention must be directed chiefly to the physical signs. Unfortunately, in view of the fact that the rational symptoms are often ambiguous, a physical exploration of the chest is apt to be neglected. The chief difficulties are encountered in distinguishing this affection from conditions in which the lung is either consolidated, retracted, or compressed by solid new growths, etc. Chief among the former is *croupous pneumonia*, and I have tabulated below the most important distinctions between it and pleurisy. The reader will be further aided by comparing Fig. 47 (which shows the physical conditions in pleurisy) with Fig. 17, on page 150, which shows the physical conditions in pneumonia.

## PLEURISY WITH EFFUSION.

## PRIMARY LOBAR PNEUMONIA.

*Rational Symptoms.*

Onset marked by chilliness persisting for a few days.	A severe rigor, lasting about one hour.
The pain is sharp, "stitch-like," and strictly localized.	Acute pain, similar, but soreness more diffused.
Cough frequent and irritating; no expectoration.	Cough accompanied by rusty or bloody expectoration.
Moderate fever of continuous type; decline by lysis.	Intense fever; decline by crisis from the fifth to the ninth day.
Systemic prostration of medium severity.	Prostration marked.
Countenance pale and anxious.	Countenance congested; mahogany flush on the cheeks.
Herpes does not appear.	Herpes quite common.

*Physical Signs.*

<i>Inspection.</i>	
Marked distention of the thorax.	None.
<i>Palpation.</i>	
Diminished or absent tactile fremitus.	Marked tactile fremitus (absent only when a bronchus is plugged).
<i>Percussion.</i>	
Flatness, with great resistance to the pleximeter-finger.	Dulness less complete, less resistance, and sometimes a tympanitic note.
Shows displacement of neighboring organs.	No displacement of neighboring organs, if uncomplicated.
If the sac be partly filled, there is a change in the line of flatness on change of position.	Absent.
<i>Auscultation.</i>	
Diminished or absent breath-sounds, bronchial breathing frequent, but diffused and distant and unaccompanied by râles, as a rule.	Harsh bronchial breathing and presence of râles in first and third stages, unless a bronchus be plugged.
Vocal resonance diminished or absent.	Bronchophony, unless a bronchus be blocked.
Friction-sound in early and late stages.	No friction-sound, except crepitant râles in the first stage.
<i>Aspiration.</i>	
Yields serum.	Yields a few drops of thick blood.

*Consolidation* of the lung, due to tuberculous infection, may be differentiated from pleurisy with effusion by means of the physical signs contrasted in the foregoing table, by the history of the case, and by the discovery of the tubercle bacillus in the sputum.

*Hydrothorax* presents physical signs that simulate strongly those of pleural effusion. Hydrothorax, however, gives the history of cardiac or renal disease, is usually bilateral, and is unassociated with a rise in temperature or with the pain or friction-sounds peculiar to pleurisy. In obscure instances some of the fluid should be withdrawn and chemically examined.

*Tumors* and *cysts* of the thorax will give complete dulness, will displace the heart, and compress the lung on the affected side, thus causing an absence of the respiratory murmur, etc. But the history of the case, the situation of the dulness (usually over the upper or middle parts of the lung), the absence of uniform distention extending to the base, and the exaggerated tactile fremitus and vocal resonance will serve to distinguish these affections from pleurisy with effusion.

*Echinococcus cyst* of the liver, or *abscess* of this organ, pushing upward, will cause retraction or even compression of the lung, and hence will also produce most of the physical signs of pleurisy with effusion. The former affections can be discriminated only by a correct appreciation of the history, by the presence not infrequently of a friction-sound on auscultation, and by the immovable, fixed upper level of dulness. If doubt remains, an exploratory puncture should be made, and the fluid withdrawn should be subjected to a chemical, microscopic, and bacteriological investigation.

An *enormous pericardial effusion* may be mistaken for a pleural effusion on the left side. In the former, however, there is commonly a history of rheumatism, and dyspnea is the most urgent symptom, while the heart-sounds are greatly enfeebled; moreover, the heart is not displaced to the right as in pleural effusion. Again, flat tympany is obtained in the posterior portion of the axilla and good pulmonary resonance at the base in the postero-lateral region of the chest, differing from the results of percussion in pleurisy with effusion.

For practical purposes it is desirable to distinguish the *tuberculous* from the rarer forms of pleurisy. This is sometimes possible by paying due regard to the previous history of the patient, including hereditary taint, by noting certain clinical peculiarities (such as associated disease of other serous membranes and of the lung and bilateral inflammation of the pleura), and by the results of an examination of the exudate. In dubious cases the guinea-pig should be inoculated with the exudate, and if the patients are tuberculous positive results may be confidently expected.

**Duration and Prognosis.**—This depends largely upon the cause. The course of acute sero-fibrinous pleurisy is not definite, but is made up of two parts—the febrile followed by the non-febrile stage. The fever lasts from one to three weeks, and is due to inflammation; it corresponds to the period when the effusion occurs, and the appearance of a non-febrile period indicates the subsidence of the inflammatory action. The effusion may be poured out rapidly, and is removed by absorption not less rapidly; more frequently, however, the effusion takes place

rather gradually, and the same is true of resolution. Again, large effusions may persist in consequence of a purely mechanical hindrance to resorption; and finally, the course may become subacute or chronic in consequence of the development of empyema. Such facts as these constitute an explanation for the great differences in the duration of the cases. Simple sero-fibrinous pleurisy has a comparatively favorable prognosis. In rare instances, however, death ensues suddenly, without adequate lesions to explain its occurrence. Moreover, the fact that a sero-fibrinous effusion may be converted into a purulent one is not to be forgotten. Again, the crippling influence upon the lung-tissue of previous attacks, owing to resulting adhesions, must be borne in mind, since chronic bronchitis and emphysema are often thus produced. Lastly, it is to be restated that most cases of pleurisy are tuberculous in origin, even though it cannot be denied that complete recovery may take place.

**Treatment.**—In the first stage the treatment is the same as for dry or plastic pleurisy. During the second stage, that of effusion, the objects of treatment are threefold: (1) To limit the extent and intensity of the inflammatory process; (2) To accomplish the removal of the effusion; and (3) To support the strength of the patient.

(1) **To Limit the Extent and Intensity of the Inflammatory Process.**—To this end two classes of agents are employed—namely, (a) *Internal*, and (b) *External*.

Among the latter are counter-irritants, as sinapisms and iodine, by means of which gentle but constant counter-irritation is to be maintained. Another agent of great worth is cold, applied by means of the ice-bag or ice-water bag, and if the temperature rises to 102° F. (38.8° C.) cool spongings of the surface of the body, together with the use of the ice-cap, will be found highly useful. Roberts recommended keeping the affected structures at complete rest by fixing in a mechanical manner the side affected. For this purpose strips of adhesive plaster must be firmly and evenly applied to the chest, and by this means the pain is relieved and the amount of inflammatory product poured out is greatly limited.

The *internal* remedies embrace quinin, the salicylates, and opium. Opium and quinin are potent in controlling inflammation of serous membranes; the former being given preferably either in the form of suppositories or hypodermically, and the latter in divided doses, in capsule, followed by a few drops of mineral acid, administering gr. xvj to xx (1.036–1.296) daily.<sup>1</sup> I have observed good results from the salicylates (3j–ij—4.0–8.0, daily), which have been warmly advocated by Fiedler, Koester,<sup>2</sup> and others, as valuable in mitigating or even aborting the inflammation of the pleuræ, and thus in limiting the amount of effusion. It must not be forgotten that the effusion is due to an inflammation, and not to a simple transudation. The use of mild diaphoretics and diuretics, together with repeated small doses of salines, also aids in reducing the inflammation in the pleura. With a subsidence of the inflammatory process the temperature falls, and, when the latter reaches a point near to the normal, our efforts should be directed toward the

<sup>1</sup> *International Clinics* (1892), vol. i., second series.

<sup>2</sup> *Annual of the Universal Medical Sciences* (1893), vol. i. (A-31).



fulfilment of the second leading indication, (2) the removal of the effusion.

Little is to be accomplished by local means, though iodine, persistently employed, sometimes does good. The following ointment may also be tried :

R<sub>x</sub>. Ung. ichthyol. (12 per cent.),  
       Ung. iodini comp.,                   *āā*. ʒvj (24.0);  
       Ung. belladonnæ,                   q. s. ad ʒij (64.0).—M.  
 Sig. Apply twice daily.

Blisters are not admissible.

Mild hydragogue cathartics, and especially the salines, after the Matthew Hay method (*i. e.* ʒij to ʒss—8.0–16.0, in the smallest possible amount of water, on rising in the morning), stimulate absorption from the pleural cavities by draining the blood of a certain amount of serum. Unirritating diuretics may also be employed, but I have found no appreciable advantage from their use. Free diaphoresis (from the use of pilocarpin) sometimes assists in the absorption of the exudate, but it should not be employed in the presence of feeble heart-action or marked displacement of the organ. Among measures to promote absorption, the best, in my own experience, is the following combination :

R<sub>x</sub>. Potassii iodidi,                   ʒj (4.0);  
       Syr. ferri iodidi,               ʒij (8.0);  
       Syr. sarsap. comp.,           ʒj (32.0);  
       Ess. pepsinæ,                   q. s. ad ʒij (64.0).—M.  
 Sig. ʒj (4.0) every four hours, diluted; the dose to be doubled at the end of four days if well borne by the stomach.<sup>1</sup>

The patient should be put upon a dry diet in order to increase the plasticity of the blood, which is thus induced to absorb the liquid exudate from the pleural cavity. The *modus operandi* of this treatment is different, but the effect aimed at is the same as when saline purgatives are given. The exudation, however, defies all efforts at removal in about 33 per cent. of the cases, and in such the withdrawal of the liquid by aspiration (thoracentesis) must be practised. The indications for thoracentesis arise at two different periods in the course of pleurisy with effusion :

(1) During the febrile stage, while efforts are being directed to combating the inflammatory process. The object during this stage is to avert imminent danger to life, and not merely to remove the fluid. The conditions demanding immediate thoracentesis are—(a) when one pleural sac is completely filled or when Skoda's resonance extends from the clavicle downward no farther than the second interspace; (b) in double pleurisies, when both sides are half filled, since death may occur from rapid filling of one or the other side; (c) in cases of copious effusions, upon the first signs of involvement of the unaffected side, such as moist râles, broncho-vesicular breathing, and impaired resonance; (d) the appearance of serious symptoms, such as orthopnea or syncopal attacks with cyanosis; (e) marked displacement of the heart, especially if one or more murmurs develop in the organ.

<sup>1</sup> The author has employed this formula in more than 50 cases with very good results.

(2) The indications for aspiration during the second or afebrile period, when the main object is to remove the exudate, are—(a) if no diminution in the quantity of liquid effusion takes place one week after the temperature has reached the normal; (b) in subacute cases, in which there is little, if any, temperature from the beginning; aspiration should not then be withheld longer than three weeks.

The operation is free from danger if carried out under antiseptic precautions and if a modern aspirator is employed. The instrument should always be tested before it is used. The patient rests in bed in the semi-recumbent posture, the arm of the affected side being brought forward with the hand placed on the opposite shoulder, so as to separate the ribs from one another. The point of puncture is in the sixth interspace on the right-hand side and the seventh interspace on the left, in the mid-axilla, or just below the outer angle of the scapula in the seventh right and eighth left interspaces, respectively. An assistant draws up the skin from the interspace, while the operator uses the fore finger of his free hand as a director. The needle should be introduced with a quick thrust, hugging the rib below the interspace, but endeavoring to avoid striking its periosteal covering. The fluid may not be obtained at the first operation, and the reasons for this failure are several. The costal pleura may be excessively thickened, or we may meet with a much-thickened fibrous band. Again, the fluid may be encapsulated; and, lastly, the needle may become blocked. Under these circumstances repeated trials should be made.

The amount of fluid withdrawn at one time should never be large (3xij to xxiv—384.0–768.0), though a relatively larger quantity may be taken during the febrile stage than during the afebrile, since in the latter instance the lung has been compressed for a longer period of time. The fluid is allowed to drain away slowly, a small needle being used, so as to invite the lung to expand in a gradual manner. If this precaution be not taken, the paretic pulmonary capillaries are apt to become the seat of sudden fresh congestion, followed by edema, and often by a speedily fatal termination. Thoracentesis is to be repeated at intervals of several days if nature does not take up the work of absorption, following the first operations. If during the operation incessant cough, dyspnea, a tendency to syncope, marked thoracic constriction, or sudden intense pain be developed, the needle must be withdrawn instantly.

Thoracentesis should not be resorted to in cases in which croupous pneumonia is associated, and never in very aged and excessively feeble persons.

(3) **To Support the Strength of the Patient.**—The powers of the system are to be maintained by a nutritious diet, bodily rest, and other hygienic measures. The lighter forms of solid food may be allowed whenever they are found to agree, and it is important to promote the digestive power, should the latter be weak, by the administration of suitable remedies. During the stage of convalescence, therefore, tonics (strychnin, quinin, and arsenic) are to be administered. The dietary should be liberal, though composed of wholesome articles. Gentle exercise in the open air is to be encouraged, and massage of the muscles of the affected side tends to re-establish their usual vigor. To bring about the best possible chest-expansion nothing is so good as light gymnastic

exercises, together with the methodical practice of deep inspirations for a minute or two at intervals of three or four hours. I am of opinion that the management of the third stage, or that of convalescence, is about the same as that of tuberculosis.

#### EMPHYEMA (PURULENT PLEURITIS).

**Definition.**—A suppurative inflammation of the pleura.

**Pathology.**—On opening the pleural sac after death we may find a thick, creamy pus, though more frequently it is sero-purulent and separated into two layers—an upper, clear, greenish-yellow serous, and a lower, thick, purulent layer. In a smaller proportion of cases the exudate is fibrino-purulent. The odor emitted from the purulent collection is either sweetish or fetid (*e. g.* when due to wounds), and, when the condition is associated with gangrene of the lung or pleura, horribly offensive. Microscopic examination shows that the inflammatory products are identical with those of purulent inflammation in general. The pleural membranes are the seat of a more intense inflammation than in acute sero-fibrinous pleurisy, and are greatly thickened (1 to 2 mm.). They present a granular suppurating surface, and both visceral and costal pleuræ exhibit perforations, and the latter, quite frequently, erosions.

Histologically, the altered membranes consist of new connective tissue, new blood-vessels, and numerous leukocytes.

**Etiology.**—The following are the chief circumstances under which empyema arises: (1) As a sequel of the acute, sero-fibrinous variety. However clear the effusion may be, it always contains corpuscular elements, which in the further progress of certain cases undergo coincident increase in numbers until the effusion presents a milky aspect, when it is said to be purulent. Thoracentesis may be responsible for this change, though never if performed under rigid aseptic precautions.

(2) In children the effusion early becomes purulent in many instances, and in some cases may be so from the start.

(3) Secondary to the acute and chronic infectious diseases (pyemia, scarlatina, pneumonia, tuberculosis, and dysentery most frequently; typhoid fever, measles, whooping-cough rarely).

(4) The disease may follow malignant affections of the thoracic organs (lungs, esophagus), or tuberculous pulmonary cavities which perforate into the pleura.

(5) Injuries to the chest may set up empyema (fracture of the ribs, stab or other penetrating wounds).

Bacteriologic investigation has shown that the organisms most frequently present are the micrococcus lanceolatus, streptococcus, staphylococcus, and tubercle bacillus. The cases due to pneumococci usually pursue a favorable course. The leptothrix pulmonalis is often found in putrid effusions.

**Clinical History.**—The symptoms vary with the cause. The onset may be characterized by acute symptoms, such as rigor, followed by high temperature and signal prostration, and in the affected side there may be severe pains, aggravated by deep breathing and bodily movements.



If the exudate becomes gangrenous, a typhoid state develops early, and the case is apt to prove fatal in the course of a few weeks. It is quite a common event for the acute symptoms that characterize the invasion to be replaced at the end of a week or more by the more obscure rational symptoms of chronic empyema. The latter, however, may develop very insidiously as a secondary affection. The rational symptoms in a well-marked case should always excite a suspicion of the presence of the affection, but cannot set the question of diagnosis at rest. The local symptoms (pain, cough, and expectoration) are of a mild character; the dyspnea, however, that is usually present may be more or less intense. I have on more than one occasion found an utter absence of these symptoms. The general symptoms are those of septic infection—diurnal chills occurring at irregular intervals, followed by great paroxysms of fever and profuse sweating—and such patients lose flesh and become pale and weak. The temperature is higher than in pleurisy with effusion, and is intermittently, though irregularly, elevated.

Peptonuria is a symptom of purulent pleurisy that is not without diagnostic value. It, however, also occurs in suppuration associated with the third stage of pulmonary tuberculosis, and in suppuration due to other causes. While not indicative of empyema, however, it serves sometimes to eliminate sero-fibrinous pleurisy. The urine also contains indican in excess in the various suppurations, at least from time to time, if not constantly. Blood-examination invariably shows leucocytosis.

If the pus is not removed artificially, it frequently breaks into the lung, penetrates it, and finally discharges through a bronchus. Pneumothorax now tends to supervene. Traube contends that necrosis of the pulmonary pleura may allow of the soaking of the pus through the spongy lung-tissue into the bronchi, without the establishment of a fistulous connection between the latter and the pleural sac, and hence without the formation of pneumothorax. Besides rupture into the lung and external rupture, empyema may perforate through neighboring organs, as the esophagus, pericardium, stomach, and peritoneum. In rare instances the pus burrows along the spine behind the peritoneum and the psoas muscle, reaching, finally, the iliac fossa and simulating psoas or lumbar abscess.

**Physical Signs.**—These are, for the greater part, identical with those of pleurisy with effusion. Attention will therefore be called only to such as are more or less distinctive of the affection. Slight edema of the chest-wall over the seat of effusion, especially in children, is often present, and if the pleural sac be not aspirated, the abscess may point externally and evacuate itself spontaneously. In the latter event a protrusion between the ribs shows itself: this may be the seat of fluctuation, and present an inflammatory appearance prior to its rupture, with subsequent discharge of its contents. The opening is usually found in the fifth interspace in front, and less frequently in the third and fourth interspaces or below the angle of the scapula behind. The upper level of the fluid does not change so readily on changing the posture of the patient, but requires a longer period of time.

Bacelli's sign, or the transmission through a serous exudate of the whispered voice, is sometimes an aid in the discrimination of pleurisy

with effusion from empyema. According to my own observation, though it is not invariably propagated by large serous exudations of the pleura, it is yet detectable in a large majority of instances, whilst I have never found it to be obtainable in chronic empyema.

**Pulsating Pleurisy.**—Pulsation synchronous with the cardiac beat in pleural effusion has received various designations (*pulsating empyema*, *empyema necessitatis*, *pulsating pleurisy*). The latter term is the most appropriate one, in view of the fact that its course takes place not only in empyema necessitatis, but also in empyema (which manifests no tendency to point externally) and rarely in sero-fibrinous pleurisy.

Its *etiology* is not definitely known. The principal causal factors, however, seem to be—(1) a copious effusion; (2) paresis of the intercostal muscles, inducing relaxation of the thoracic wall; (3) a somewhat forcible heart-beat (Henry). The rational *symptoms* of empyema are present. The physical signs are also identical with those of the latter affection, with the pulsation superadded. There are instances in which palpation alone detects the systolic pulse in the pleural effusion. With rare exceptions the effusion occupies the left pleural sac. The pulsation may be limited to two or three interspaces or it may be visible over the entire antero-lateral aspect of the chest; pulsation at the back, however, is rare.

**Differential Diagnosis.**—An absolute distinction between empyema and *pleurisy with effusion* rests solely upon the results of exploratory puncture. For this purpose the needle attached to the ordinary hypodermic syringe, or, preferably, the surgeon's exploring needle, may be employed, withdrawing but a very small quantity of the fluid, which should be examined both macroscopically and microscopically.

Pulsating pleural effusion simulates closely *aneurysm of the thoracic aorta*. When pulsation occurs in empyema, however, it is seen to be to the left of the normal course of the aorta: the rational symptoms and usual physical signs of purulent pleural effusion are usually present also, while the vascular symptoms and signs of aneurysm of the aorta (thrill, bruit) are absent.

**Prognosis.**—Empyema is a serious disease, but, obviously, the outlook will be modified by the special etiology. Spontaneous absorption may occur, though it is extremely rare. The discharge of the contents of the pleural sac through the bronchial tubes is a comparatively favorable event, some cases in which this occurs recovering, while in others death follows in consequence of the sudden inundation of the bronchi. An empyema may, in rarer cases, empty itself externally with favorable issue (*empyema necessitatis*). Evacuation of the pleural cavity is often followed by a continuous discharge of pus for an indefinite period, but the pus cannot be allowed to remain within the thoracic cavity with impunity. As a result of the long-continued suppurative process, death may take place by slow asthenia. It must not be forgotten, however, that an unfavorable termination may be, in part at least, ascribable to certain associated affections (phthisis, pericarditis).

Among children the outlook is much more favorable than among adults. The prognosis has, however, been rendered less serious by the application of surgical principles in the treatment of the disease. In all cases in which recovery ensues there is a progressive obliteration of



the pleural cavity, owing to adhesions, which finally become universal and lead to marked retraction of the affected side.

**Treatment.**—The treatment of empyema is chiefly surgical. In a child the condition may terminate in recovery without operation, and hence may, at this period of life, be allowed to run for two or three weeks, thoracentesis being resorted to if suffocation be threatened. In an adult, however, if the purulent effusion be copious, aspiration should be performed at once as a temporary means of relief. Empyema following pneumonia may terminate favorably after one or more tapplings; but unless contraindicated by an unfavorable general condition of the patient, such as is met with in the closing stages of pulmonary tuberculosis, free incision should be made without delay. The pleural sac should be opened in the fifth or sixth interspace to the left of the mammary line, the incision being from 2 to 3 cm. in length. Resection of a rib is advocated by most surgeons, but if the drainage afforded by free incision be complete resection is unnecessary. It is only indicated when, by approximation of the ribs, the free exit of the pus is hindered (Verebeyli<sup>1</sup>). Opinions are divided as regards the value of irrigation of the pleural cavity. When the pus emits an offensive odor irrigation with a disinfecting solution is imperative. Carbolic acid should, however, not be used. In rare instances accidents arise during irrigation (sudden collapse, convulsions), and I have repeatedly observed a dangerous, and in one instance a fatal, collapse as the result of irrigation in children. The careful insertion of a roll of iodoform gauze is a method to be preferred to irrigation, except when the effusion is stinking. For further details in the operative treatment of empyema the reader is referred to text-books on surgery. Every effort should be made to favor obliteration of the cavity during post-operative treatment. The indication is to bring about the best possible degree of re-expansion of the compressed lung, and in order to accomplish this the method advised by Ralston James has been practised with great success in the surgical wards of the Johns Hopkins Hospital. The patient daily for a certain length of time, increasing gradually with the increase of his strength, transfers water by air-pressure from one bottle to another. The bottles should be large, holding at least a gallon each, and by an arrangement of tubes, as in the Wolff bottle, an expiratory effort of the patient forces the water from one bottle into the other. In this way expansion of the compressed lung is systematically practised. The abscess-cavity is gradually closed, partly by the falling in of the chest-wall and partly by the expansion of the lung.<sup>2</sup> In long-standing cases, in which the lung cannot expand on account of thick bands of adhesion, the pleural layers cannot be brought into juxtaposition without more or less sinking in of the chest-wall. This retraction of the thorax is probably hastened by timely resection of one or more ribs, the amount of bone to be removed depending upon the "expansive power of the lung and elasticity of the thorax."

The duration of empyema is longer than in pleurisy with effusion, and the former affection tends to exhaust to a greater degree the powers of the system than the latter; hence the physician's attention should be

<sup>1</sup> Quoted in *Annual of the Universal Med. Sciences*, 1892, vol. i. sec. A.

<sup>2</sup> Osler's *Text-book of Medicine*, p. 605.



directed chiefly to the support of the vital forces, modified to some extent by the special etiology in the individual cases.

#### CHRONIC PLEURISY (CHRONIC ADHESIVE PLEURISY).

**Definition.**—Chronic inflammation of the pleural layers—(a) with effusion, and (b) without effusion.

(a) **Chronic Pleurisy with Effusion.**—This sub-variety may follow acute sero-fibrinous pleurisy, and less frequently it has an insidious development. The morbid lesions, including the character of the exudate, may also be identical with those of the acute or subacute forms of the affection. Fibrin and serum are present in varying relative proportions, the latter, however, as a rule, in preponderating proportion when compared with the composition of the exudate in acute pleurisy. The secondary consequences of copious acute effusions also are met with—*i. e.* displacement of adjacent organs (liver, spleen, heart) and unilateral dilatation of the chest. When the fluid is either absorbed or removed and the case ends in recovery, marked contraction of the affected side results, since the lung, which is covered by thick, organized bands of adhesion, cannot re-expand. *Symptoms.*—But for slight dyspnea upon muscular exercise the subjective symptoms are frequently wanting. The pulse is compressible and accelerated, as a rule, and there is a trifling rise of temperature in the evening hours. If the effusion becomes purulent, hectic fever develops, leading to asthenia, and the latter condition eventually terminates life. Death may also be due to secondary suppurations (abscess of brain, etc.). In most cases occurring in children the effusion early changes to pus. The physical signs do not differ from those in acute sero-fibrinous pleurisy. The *duration* of the cases varies from three months to several years, or intercurrent pulmonary tuberculosis may shorten the course of the affection.

(b) **Chronic Dry or Adhesive Pleurisy.**—(1) This may succeed to the acute or chronic sero-fibrinous pleurisy. If the liquid portion of the exudate is absorbed, the pleural membranes come into more or less close apposition, being separated only by fibrinous elements that become organized into a layer of firm connective tissue. Hence the two layers of the pleura, that are greatly thickened, cannot be separated, owing to the firmness of the adhesions. Most frequently the autopsy shows the latter condition to be most pronounced at the base, while the lung is found to be compressed and the seat of fibroid change. If it follows the acute form, the extent of retraction is slight, since there are no dense fibrous bands to prevent a fair degree of lung-expansion: if it succeed the chronic form, however, or empyema, the extent of retraction and flattening will be quite marked. The exudate may undergo calcareous degeneration, and occasionally little pouches of fluid may be found between the false bands.

There is a large class of cases that are dry from the onset (*idiopathic dry chronic pleurisy*), and this variety may either be a sequel of acute plastic pleurisy or primarily tuberculous. The condition is very commonly met with at autopsy in subjects who during life had never presented symptoms of pleurisy with effusion. The plastic exudate, however slight, invariably tends to become organized, with result-

ing fibrinous adhesion of the two layers of the pleura. Most generally the adhesions are circumscribed, and if tuberculous in origin are most frequently apical and often bilateral. Under these circumstances small caseous masses and little tubercles may be found embodied in the somewhat thickened pleura. General synechia is, however, not rare, particularly unilateral.

*Symptoms.*—Definite rational symptoms are rarely present, and the physical signs lack uniformity or may be entirely negative. In other cases of a mild grade the main characteristics are restrained mobility of the affected side and feebleness of the respiratory murmur. In rarer cases the weakness of the breath-sounds is out of all proportion to the expansive motion of the chest. In still another category—composed of a considerable number of instances—certain physical signs are quite pronounced. Inspection reveals decided contraction, with immobility of the affected side and a compensatory distention of the healthy side. The heart is displaced, and the apex-beat may be missing (*e. g.* when the heart is drawn or pushed behind the sternum, or overlapped by the emphysematous lung). The spinal column is curved, the scapula dislocated, the shoulder ill-shapen and drooping, and the lower part of the thorax shrunken, while the ribs are obliquely placed and closely approximated, or even overlap one another. The tactile fremitus is decreased or absent over the lower portion of the chest, and there is impaired percussion-resonance or dulness over the same area. The breath-sounds on auscultation are exceedingly feeble, and in some instances an occasional dry, leathery, or creaking friction-sound is audible.

Rarely, and particularly if the case be tuberculous, vasomotor symptoms arise in chronic pleurisy, such as unilateral flushing or sweating of the face, or dilatation of the pupil.

Doubtless some of the instances belonging to this affection merge into the pleurogenous type of cirrhosis of the lung, and fatal complicating conditions may arise in connection with the general circulation. Thus I have observed in one instance enlargement followed by dilatation of the right ventricle, and in turn by general dropsy, with fatal result.

**Treatment.**—In the treatment of this affection two objects must receive especial attention: (1) the removal of any effusion that may be present; and (2) the improvement of the nutrition of the patient. The first indication is presented only by a limited number of the cases, and the rules for meeting it have been stated in the treatment of sero-fibrinous pleurisy and empyema; the second indication is presented by all cases. Careful regulation of the diet is of the utmost importance: it must be generous, with modifications to suit special diatheses (as the gouty or tuberculous), if they be present. Lung-gymnastics are most useful if methodically pursued. The method of Ralston James (previously described) richly deserves a trial in suitable cases. It is to be borne in mind, however, that in old cases efforts at overcoming the lung-pressure will be unsuccessful. Climato-therapy is advantageous for this class of sufferers, particularly if the slightest tendency toward tuberculosis exists; and in my own experience low, mountainous elevations combined with purity of atmosphere have given the best results. Of medicines little need be said. It is especially important to promote

the digestive power of the patient to the greatest possible extent. In cases in which the digestive function has been feeble I have observed excellent results from a brief stay at any well-regulated seaside resort or in the country. We may also try, with a probability that the effect will be beneficial, small doses (ʒj—4.0) of cod-liver oil, three times daily after meals, or the following formula:

Ry. Acidi muriat. dil.,	ʒijss (10.0);
Pepsini pur.,	ʒij (8.0);
Tinct. nucis vom.,	ʒiiss (6.0);
Glycerini,	ʒiiss (48.0);
Aquæ,	q. s. ad ʒij (64.0).—M.

Sig. ʒj (4.0), well diluted, ten minutes after each meal.

Intercurrent catarrh of the stomach may sooner or later become a troublesome feature, and in combating it lavage is frequently our most effective measure.

## PNEUMOTHORAX.

(*Sero-pneumothorax*; *Pyo-pneumothorax*.)

**Definition.**—A collection of air in the pleural cavity. Since the latter, as a rule, contains at the same time serum or pus, the terms sero- and pyo-pneumothorax are frequently employed to describe the same condition.

**Pathology.**—When the pleural sac is punctured air usually escapes, accompanied sometimes by an audible hissing sound. The pleural sac in pure pneumothorax is greatly distended, and the lung is impacted against the spinal column. Other organs (spleen, heart) are also displaced, owing to positive intrathoracic pressure. The heart is not dilated, however, and the relation of its parts is maintained much as in the normal condition (Osler). The air may occupy but a portion of the pleural cavity, on account of previous firm adhesions (*circumscribed pneumothorax*). The point of perforation, as a rule, can be easily found, and most frequently corresponds to the seat of rupture of the tuberculous cavity or superficial caseous mass. In other instances the cause of pneumothorax cannot be discovered. Inflation of the lung under water may reveal the aperture, which is usually quite small, by the escape of air-bubbles at the seat of puncture. Occasionally a fistulous connection between the pleural sac and the bronchi can be readily traced.

*Simple pneumothorax* is, however, of rare occurrence. The air that gains admission into the pleural sac is laden with micro-organisms, which set up various forms of inflammation, accompanied by equally various exudations. Hence the cavity is usually filled, in part, with an effusion that is purulent or sero-purulent, as a rule, and rarely serous or sero-fibrinous.

**Etiology.**—There are both *predisposing* and *exciting* causal influences, and among the former are—(a) *age*; the condition occurring in



adults as a rule, though instances are also observed in young children; (b) *sex*; males suffer more often than females; (c) the left side is affected nearly twice as often as the right; (d) *emphysema*, in which the superficial air-sacs are dilated and atrophied, rendering the latter liable to rupture from excessive muscular exertion.

The **exciting** causes are—(1) *Perforation of the lung and pulmonary pleura* (the most frequent cause), arising in one or other of three ways—(a) From the rupture of a tuberculous cavity into the pleural cavity. This accident rarely occurs at the apex of the lung, but commonly near the upper border of the lower or middle lobe; and less frequently near the lower border of the upper lobe. A caseous focus immediately beneath the pleura may also, during the process of softening, puncture the pleural sac and invite the entrance of air. From this cause we sometimes see pneumothorax developing during a very early stage of pulmonary tuberculosis. It cannot occur, however, except in cases in which previous adhesions have failed to form at the point of perforation. (b) As the result of necrotic processes, in connection with certain other lung-affections, as gangrene, broncho-pneumonia, suppurating bronchial glands, abscess, and echinococcus cysts. (c) From rupture of the normal air-sacs in consequence of severe muscular effort (S. West, DeH. Hall). This accident is sometimes ascribable to the violent paroxysms of cough in pertussis.

(2) Some cases of *empyema*, by perforating the visceral pleura, the lungs, and bronchi.

(3) Perforations of the pleura in *malignant disease* and *abscess of the esophagus*.

(4) A *peripheral bronchiectasis* may open the pleural space and thus establish a communication between it and a bronchus.

(5) Pyo-pneumothorax may be of *subdiaphragmatic origin*, consecutive to *perforation by malignant disease* or *ulcer of the stomach or colon*.

(6) Pneumothorax may be occasioned by *gases* resulting from the decomposition of a pleural exudate.

(7) *Wounds* causing direct or indirect perforative lesions of the lungs. Fractures of the ribs may produce laceration of the visceral pleura, and afford an opportunity for the ingress of air into the pleural sac.

**Symptoms.**—The earliest symptoms vary according to the cause or causes that produce the condition. When it develops, as it does so often, in the course of pulmonary tuberculosis, the first symptom is a sudden agonizing pain in the side, accompanied by marked dyspnea and frequently cyanosis. The dyspnea is often accompanied by a sense of impending suffocation. The severity of the pain and the degree of oppression depend largely, however, upon the amount of air that gains entrance into the pleural sac, the rapidity with which the air escapes into the pleural cavity, and the condition of the latter as regards the presence or absence of previous pleuritic adhesions. If the orifice be large and valvular, the air cannot escape, but rapidly accumulates and forces all the air out of the lung by compression; the patient then sinks rapidly into collapse from shock, and sudden death ensues. Fortunately, the latter event is rare. The respirations are frequent (60 or more per minute); the pulse is also frequent and feeble, sometimes reduced to a

thread; and cold sweats are not uncommon. The temperature at first is apt to fall one or two degrees below the normal, owing to sudden collapse; fever, however, follows almost invariably, and frequently is of the hectic type. Its cause is pleuritis, often of a purulent type, and if this be the case the dyspnea may be due in part to the increased effusion. The patient now also suffers from the grave symptoms of empyema above described. Edema of the hand of the affected side is sometimes present

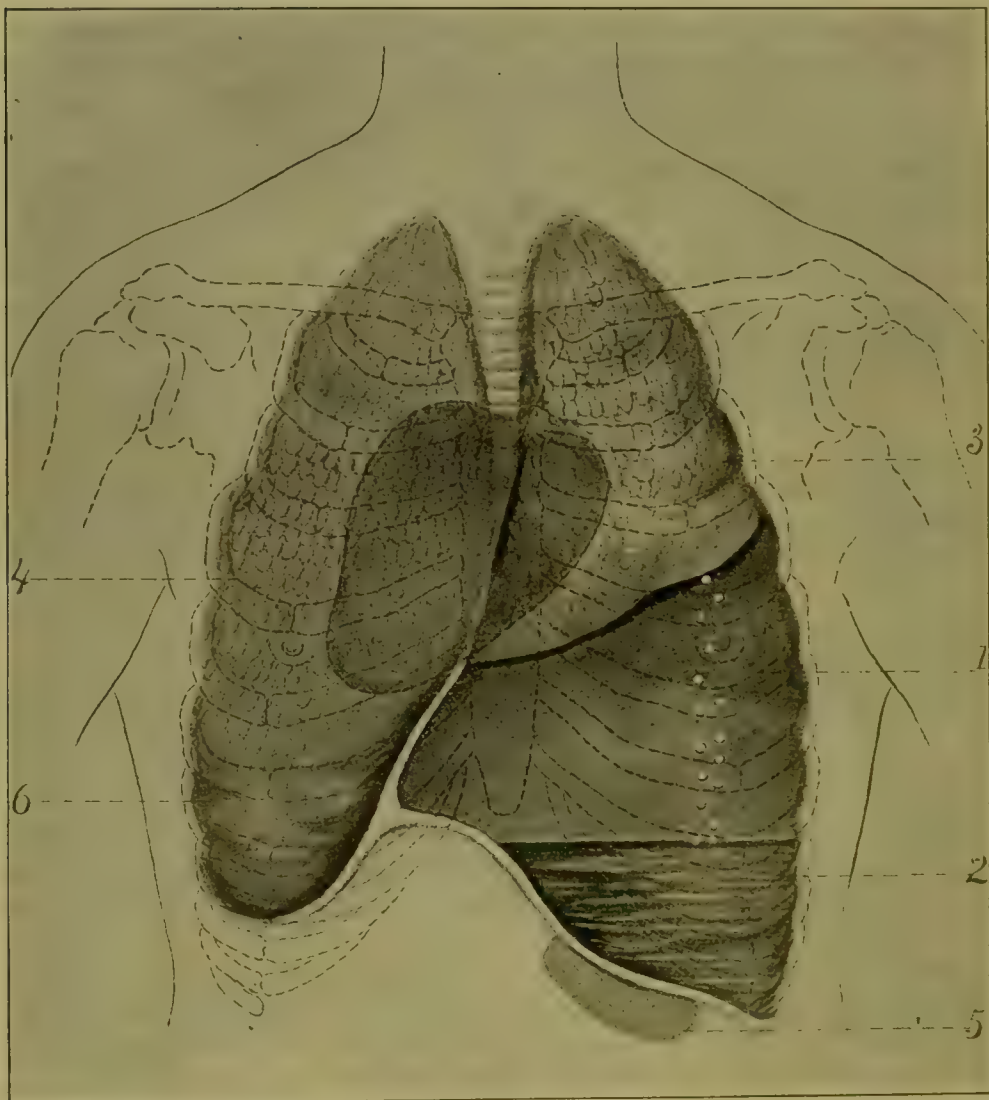


FIG. 48.—1. Air in the pleural sac; 2. fluid exudate at base of pleural sac; 3. compressed portion of lung; 4. displaced heart; 5. depressed spleen; 6. mediastinum pushed toward the right.

as an early manifestation, and, as a rule, rapidly disappears (Weil). When pneumothorax develops in the last stages of phthisis acute symptoms are often entirely absent.

**Physical Signs.**—These are marked (see Fig. 48). *Inspection* shows marked distention and immobility of the affected side; also some degree of distention with unnatural mobility of the healthy side.

*Palpation* shows the tactile fremitus to be diminished above and greatly diminished or wholly absent over the effusion below. Edema of

the chest-wall can frequently be made out. The impulse-beat of the heart is found to be feeble and displaced.

On *percussion* a modified tympanitic note (*bell-tympany*) can usually be elicited over the area corresponding to the contained air, and the excessive tension in the pleural sac, due to the enormous amount of air contained therein, causes an elevation in the pitch of the percussion-note even to dulness. The "cracked-pot sound" is audible when the air in the pleural cavity freely communicates with the external air. Wintrich's sign, or a change in the pitch of the percussion-sound when the mouth is open or closed (being raised when the mouth is closed and lowered when open), may also be observed. In pyo-pneumothorax a flat note is elicited from the base upward as far as the fluid extends, and there is a more marked change in the upper level of flatness than in simple pleurisy, on changing the posture of the patient. Modifications in the pitch of the percussion-sound result from an alteration in the form as well as in the dimensions of the air-space. Owing to displacement of the heart, there is, as a rule, resonance over the normal cardiac region, and particularly when the patient assumes a recumbent posture. The liver and spleen, according to the side affected, are displaced downward to a greater degree than in simple pleural effusion.

*Auscultation* discloses a greatly weakened or altogether suppressed respiratory murmur when collapse of the lung is incomplete. Not infrequently amphoric breathing is audible and bronchial râles possessing a metallic quality are sometimes heard, as well as metallic tinkling on deep inspiration or on coughing. The metallic tinkling is caused frequently by drops of fluid falling from above upon the surface of the effusion; less frequently by a re-echoing of vibrations of moist bronchial râles communicated to the air in the pleural chamber. The vocal resonance is enfeebled, as a rule, and evinces the same metallic quality. The so-called coin test is a pathognomonic sign, and is elicited in the following manner: An assistant places one coin on the front of the chest and taps it with another while the ear is placed on the thorax posteriorly, where will be heard the intensified echo of the coin-sound thus produced. Another most characteristic sign is the so-called Hippocratic succussion, which is elicited by placing one ear upon the patient's chest while the latter's body is shaken, and a distinct splashing sound is heard.

**Diagnosis.**—When the attack is of ordinary severity, pneumothorax is diagnosticated by the history of one or other of the causal factors, together with certain physical signs that do not belong to any other affection (*coin-sound*, *succussion-splash*). It is only when the air and fluid in the pleural sac are encapsulated that it may become difficult to eliminate (*a*) a large pulmonary cavity; (*b*) excessive gaseous distention of the stomach; (*c*) an abscess below the diaphragm into which air has entered (*pyo-pneumothorax subphrenicus*); (*d*) a diaphragmatic hernia; (*e*) emphysema; and (*f*) pleurisy with effusion.

(*a*) *A Large Pulmonary Cavity.*—The "cracked-pot sound" and Wintrich's sign are more frequent in cavity than in pneumothorax, and the former condition does not tend to dislocate the adjacent organs. There is no response to the coin test and an absence of the succussion-



splash; both of which signs are often present, even in circumscribed pyo-pneumothorax. Tabulated, these points of difference are—

PYO-PNEUMOTHORAX.

Immobility and bulging of the interspaces. The apex-beat is usually displaced.  
 Diminished vocal fremitus.  
 Tympanitic percussion-note. The effusion sinks to the base, and yields dullness, the outline of which changes with the posture of the patient.  
 Respiratory murmur and vocal resonance usually absent. Amphoric breathing may be heard if the opening in the lung is patulous. Bell-tympany and Hippocratic succussion-splash are noted.

LARGE PULMONARY CAVITY.

Immobility, flattening of the chest, and depression of the interspaces. Apex-beat not displaced.  
 Fremitus usually increased.  
 Percussion gives tympany or a "cracked-pot sound," and Wintrich's change of sound as a rule.  
 Bronchial breathing is heard, and the vocal resonance is increased. Crackling, gurgling râles, cavernous or amphoric breathing, and pectoriloquy may be present. Absence of bell-tympany and succussion-splash.

(b) The possibility of *excessive gaseous distention of the stomach* is to be eliminated by the history of the case and by the happy results afforded by the application of the therapeutic test, evacuation of the stomach and bowels.

(c) *Subphrenic Abscess containing Air*.—This is exceedingly rare, and occurs relatively oftener on the right than on the left side (Leyden). Its leading causes are ulcers of the stomach or duodenum, followed by circumscribed peritonitis, perforation, and abscess, the latter occupying a position immediately beneath the diaphragm and above the liver. The gases that gain admission to the abscess-sac from the intestines force the diaphragm upward, and thus cause retraction or even compression of the lung. The symptoms and signs are now identical with those of circumscribed pyo-pneumothorax, limited to the base; but a knowledge of the steps in the production of subphrenic abscess, the symptoms and history of ulcer of the stomach or intestines, succeeded first by peritonitis and then by symptoms of pyo-pneumothorax on the one hand, and a knowledge of the etiology of pneumothorax on the other, should lead to a correct inference.

(d) *Diaphragmatic Hernia*.—This either results from a severe injury or is congenital, and the most valuable point of difference between hernia of the diaphragm and pneumothorax is the peculiar cause of the former. The next most valuable point is the fact that the hernial protrusion may return suddenly to its normal position, whereupon the patient will be relieved; the condition may then reappear not less suddenly. The third distinctive feature is the presence of rumbling sounds in the protruded bowel. All other signs and symptoms of one affection may have their counterparts in those of the other.

(e) Pneumothorax may be confounded with *emphysema* by the careless observer; but the latter affection is slow in onset, free from serious shock, is bilateral as a rule, and does not exhibit the distinctive physical signs of pneumothorax (metallic tinkling, coin-sound, succussion-splash). In *pleurisy with effusion* hyper-resonance may be noted above the fluid, but it lacks the bell-like tympany of pneumothorax. Over the same area there is diffuse, distant, bronchial breathing (at times slightly amphoric), whilst the metallic tinkling, coin-sound, and succussion-splash are totally wanting.

**Prognosis.**—This depends solely upon the cause. The cases attributed to advanced phthisis usually reach a fatal issue in the course of one, two, or more weeks, and rarely they run a very rapid and fatal course. On the other hand, the pulmonary condition seems to be favorably influenced by its occurrence. Following empyema, pneumothorax sometimes takes a favorable course. It is fraught with especial danger when it is the resultant condition of some acute lung-disease (gangrene, abscess, broncho-pneumonia).

**Treatment.**—The leading indication is the alleviation of the patient's sufferings by a prompt resort to morphin, and it often becomes necessary to administer it hypodermically. If the patient's previous strength has been moderately good, the question of operative interference should be seriously considered, the nature of the surgical procedure then depending upon the character of the effusion. If this be sero-fibrinous, aspiration, as in simple pleurisy, must be performed to relieve the urgent dyspnea and the embarrassed cardio-pulmonary circulation; if purulent, permanent drainage should be procured for the same indications. When pneumothorax develops late in phthisis, radical measures are not to be thought of, and the physician must rely upon aspiration (when necessary) to oppose urgent symptoms. We may also tap the air-chamber above the fluid with a fine needle, with a view to lessening the excessive tension. Unverricht has recently reported good results from a somewhat novel mode of treatment. When there is a pulmonary fistula present, he inserts a tube into the pleural sac. This allows free entrance of air, the lung collapses completely, and the fistula has a chance to heal. For the dyspnea, atropin administered hypodermically is valuable; for the feeble cardiac action, alcoholic stimulants, aromatic spirits of ammonia, strychnin, ether, and other cardiac stimulants should be employed. Locally, cutaneous irritants may be applied (turpentine stupes, mustard pastes).

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## HYDROTHORAX.

*(Dropsy of the Pleura; Thoracic Dropsy).*

**Definition.**—A collection of transuded serum in the pleural cavity.

**Pathology.**—Hydrothorax is, as a rule, a bilateral condition. The transudate is a clear, amber-colored liquid that is free from fibrin, but may contain cholesterin and a few endothelial cells. It has an alkaline reaction, a comparatively low specific gravity (1009 to 1012), and is non-inflammatory. The pleural surfaces are usually smooth, though sometimes decidedly pale and edematous. The mechanical effects of hydrothorax upon the lungs and other thoracic and abdominal viscera are similar to those of the exudates that accompany inflammation of the pleura, though they are rarely so marked as in sero-fibrinous pleurisy.

**Etiology.**—Hydrothorax is a secondary affection, and is usually connected with one or other of the various forms of general dropsy (hemic, renal, cardiac). The cases that are due to blood-impoverish-

ment are more numerous than is generally indicated by writers upon the subject, and not infrequently is hydrothorax symptomatic of either chronic dysentery, chronic diarrhea, leukemia, pernicious anemia, carcinoma, malaria, syphilis, or scurvy. Strictly local causes may also induce it, as carcinoma of the pleura, or the compression of the superior vena cava or of the thoracic duct by a tumor.

**Symptoms.**—The subjective symptoms are attributable to the mechanical effects of the fluid, and the causal affection may have symptoms quite in common; these are dyspnea (often culminating in orthopnea), cyanosis, asthmatic seizures, and a feeble circulation. The general symptoms arise from the primary affection.

**Physical Signs.**—The physical signs are much the same as in pleurisy with effusion—with this difference, that they are more often present on both sides of the chest. Hydrothorax is often unilateral, however, and an enlarged right auricle may be the cause of this condition in some instances. The right side is the one usually affected. I have also observed that quite frequently the two sides of the chest exhibit great variations as to the relative amount of fluid contained.<sup>1</sup>

**Prognosis.**—This depends upon the nature of the primary disorder that causes the dropsical transudation.

**Treatment.**—The treatment of hydrothorax has intimate relations with the indications presented by the underlying affection. If the measures directed toward the removal of the general dropsy, of which hydrothorax is a part, are unsuccessful, and the amount of transudation in the pleural sac interferes with the functions of the heart and lungs, then aspiration must not be too long delayed, and must be repeated as often as occasion demands.

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## NEW GROWTHS OF THE PLEURA.

ALMOST all instances of new growths developing in the pleura are secondary to primary carcinoma of the lung, the pleura being invaded by the direct extension of the neoplasm. It may also arise by metastasis from carcinoma of the lung, mammary glands, etc. The pleura presents circumscribed areas of thickening, or the growth takes the form of papular projections from its surface, and as these enlarge they become pedunculated. Their size varies from that of a pea to that of an orange. The adjacent pleura is inflamed, often adherent, and much thickened, and an effusion into the pleural cavity is often observed.

*Primary carcinoma* of the pleura is very rare indeed, and E. Wagner, who first described it, called it endothelial carcinoma. Most pathologists of to-day, however, look upon endothelioma as a variety of *sarcoma*. It owes its origin to a proliferation of the endothelial cells of the connective tissue and the lymph-apparatus of the pleura. This invariably assumes the diffuse form, and by metastasis we have involvement of the other organs (lungs, lymphatics, liver).

Spindle-cell sarcoma of the pleura, as well as the round-cell variety, is occasionally met with.

<sup>1</sup> For the differential diagnosis between pleurisy and hydrothorax see Pleurisy, p. 552.



**Symptoms.**—The subjective symptoms are slight in cases in which there is a single circumscribed carcinomatous mass in the pleura; but they are quite severe in the diffuse form, particularly when, as commonly occurs, it is of a secondary nature. The symptoms are now those of plastic or sero-fibrinous pleurisy, in addition to those of primary carcinoma of the lung, and the former may oftentimes more or less completely overshadow the latter.

**Diagnosis.**—The circumstances under which the condition arises often throw the strongest light upon its nature. The symptoms of slowly developing pleurisy, either plastic or sero-fibrinous, following carcinoma of the lung or the breast, and accompanied by the cancerous cachexia, would point strongly to the existence of *carcinoma of the pleura*. Characteristic cancerous elements may also be found by microscopic examination of the fluid obtained on exploratory puncture, and this should never be neglected in suspected instances.

The difficulties surrounding the diagnosis of primary carcinoma of the pleura are great and usually insurmountable. The cases are very similar in their clinical manifestations to *chronic pleurisy with or without effusion*. Pain is always a more prominent symptom, however, than in simple chronic pleurisy, and this fact, when combined with evidences of a cancerous cachexia, should excite strong suspicions.

The prognosis is wholly unfavorable, and the treatment merely palliative.

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## DISEASES OF THE MEDIASTINUM.

THE affections of the mediastinum may be divided into four classes: (a) Inflammation, (b) Tumors, (c) Diseases of the thymus gland, and (d) Mediastinal hemorrhage.

(a) **Inflammation.**—This may affect (1) the glands or (2) the connective tissue. **Lymphadenitis** of moderate grade is found in association with broncho-pneumonia and the various forms of bronchitis. The condition appears in its most pronounced form in the bronchitis of measles, influenza, and whooping-cough, and De Mussy held that enlargement of the glands in the posterior mediastinum is potent in exciting paroxysms of whooping-cough. According to De Mussy and Guitéras, these glands when greatly enlarged give rise to dulness in the upper part of the interscapular region or down to the fourth dorsal vertebra in cases of influenza and whooping-cough. I have, moreover, been able to confirm this dictum in cases of influenza, though aware of the fact that many authorities consider it questionable. Tuberculous lymphadenitis is elsewhere described (*vide* Tuberculosis, page 271). The mediastinal lymph-glands may undergo suppuration in consequence of local specific infection, and, though not recognizable during life, it should be recollected that the condition may lead to perforation into either the esophagus or a bronchus, with serious results. In other instances spontaneous absorption occurs, leaving behind inspissated contents that undergo calcareous change.

**Abscess of the Mediastinum.**—This is of rare occurrence, its most frequent seat being the anterior mediastinum. Of the commoner causes may be mentioned traumatism and the infectious diseases—erysipelas, rheumatism, measles, and small-pox in particular. It may also be the result of an extension of a suppurative process from neighboring structures. Pulmonary tuberculosis is the most potent factor in producing chronic abscess in this situation.

**Symptoms.**—*Acute Abscess.*—Pain and tenderness in the sternum are the most prominent features, the pain being acute and often of a throbbing character. Cough and dyspnea are usually present. The general features are fever, frequently accompanied by rigors and profuse sweats and considerable physical prostration. The chief physical sign is dulness upon *percussion*, usually found anteriorly and increasing gradually with the development of the abscess. Later, the tumor may reach the surface of the body, and rarely the sternum is eroded. *Palpation* now detects pulsation and fluctuation. The abscess may either find its way downward into the abdomen, or it may perforate the trachea or the esophagus.

In *chronic abscess* the symptoms bear a closer similarity to those of solid tumors than those in the acute form. Fortunately, chronic abscess quite often results in spontaneous cure, in which case it is in part absorbed, and the remainder of its contents become inspissated. In obscure cases an exploratory puncture with a small needle may be safely practised, and with definite results, as a rule.

**Diagnosis.**—Abscess must be differentiated from solid mediastinal tumors and aneurysm. The more acute onset and general symptoms of the suppurative process (hectic type of fever, chills, sweats) and the more rapid course will serve to distinguish abscess from *aneurysm* on the one hand, and *solid tumors* on the other. Further, the absence of strong expansile pulsation, diastolic shock, and the aneurysmal bruit aid materially in eliminating aneurysm of the arch.

The **treatment** is mainly surgical.

(b) **Tumors of the Mediastinum.**—Two forms only demand practical consideration—carcinoma and sarcoma. Hare's analysis of 520 cases gave the following ratio: of carcinoma, 134; sarcoma, 98; lymphoma, 21; fibroma, 7; dermoid cyst, 11; hydatid cyst, 8; and fewer cases of ecchondroma, lipoma, and gumma. In 48 of the cases of carcinoma and in 33 of sarcoma the tumor occupied only the anterior mediastinum. It is quite certain, however, that sarcoma, and not carcinoma, is the commoner neoplasm of this region. The clinical term "cancer" was formerly used promiscuously by many authors, and the pathologic diagnosis was then difficult, so that statistics are notoriously fallacious. Upon investigating 25 of the older reports of "cancer," Pepper and Stengel found in 13 unquestionable evidence that the growth was sarcoma, while in the remaining 12 they could not, for the greater part, decide to which form the disease belonged. Primary sarcoma may spring from the remnant of the thymus gland, from the lymphatic glands, the pleura, or lungs, or from the fibrous tissues of the mediastinum. Primary carcinoma may originate in the esophagus, bronchi, lungs, or rarely in the thymus gland. Secondary mediastinal tumors are most apt to have their seat in the lymphatic glands. Carcinoma is less frequently primary than sar-



coma. Among *predisposing causes* are sex and age—males being more prone to the affection than females, and the period of chief liability is between the thirtieth and fortieth years.

**Symptoms.**—The earlier symptoms are quite indefinite. The patient complains of slight substernal pains, slight dyspnea, and general languor. Later, with the slow increase in the size of the tumor, pressure-symptoms gradually become more pronounced.

The pain may or may not be severe, but is invariably accompanied by a feeling of oppression. Its chief seat is in the upper sternal region, but it may radiate to the sides of the chest and even down the arms (in which case it is due to pressure on the brachial plexus). Dyspnea appears early, is constant, and may become most intense. It is caused by pressure either upon the trachea, upon a primary bronchus, or upon a recurrent laryngeal nerve. Asthmatic seizures may occur before there is constant dyspnea and before the tumor has reached notable size within the chest. Less frequently, and to a less extent, the dyspnea is dependent upon dislocation of the heart or upon accompanying hydrothorax due to venous stasis. There is cough, which may be paroxysmal and of a brazen character, and as in aneurysm it may manifest implication of the recurrent laryngeal; for a like reason aphonia may be present. There may be dysphagia from pressure upon the esophagus, though this is rare. If, as may happen, there is an inflammation of the vagus or sympathetic nerve, the rate of the pulse may be affected, and the latter be either slowed or markedly quickened as a result. Owing to implication of the sympathetic there may be local hyperemias and pupillary changes, and particularly an inequality of the pupils. Rarely, by making external pressure upon the sternum, dilatation of the pupil may be produced.

*Compression of the superior vena cava or of the subclavian vein* may be followed by cyanosis and edema of the parts drained by these vessels, and the early occurrence of venous occlusion and marked dilatation of the superficial veins is quite characteristic. But if the degree of pressure increase slowly, collateral circulation may be established completely. Less frequently the inferior cava may also be pressed upon.

**Physical Signs.**—*Inspection.*—In advanced cases a swelling, usually somewhat irregular and often diffuse, appears in the sternal region. The tumor may cause erosion of the sternum, and a little later occupy a position immediately beneath the skin, Osler<sup>1</sup> being of the opinion that the rapidly-growing lymphoid tumors, more commonly than others, perforate the chest-wall. I saw a case in which the perforation occurred at the right edge of the sternum, precisely at the point at which aneurysms of the ascending arch most frequently appear. In the early stages, however, this prominence is not present. *Palpation.*—When a tumor is present it may pulsate distinctly, and the heart's apical impulse may be detected in various abnormal positions. Tactile fremitus is absent over the seat of the growth if the latter be in contact with the chest-wall.

On *percussion* dulness is noted, and this is true even in many instances that do not present a visible swelling. The dull area varies in outline with the size and position of the tumor. *Auscultation* usually reveals

<sup>1</sup> *Practice of Medicine*, p. 579.



no sounds over the dull area, except a bruit in rare instances. The heart-sounds are inaudible over the tumor-site as a rule, and the breath-sounds and vocal resonance are feeble or absent. To the above physical signs are frequently added those of pleural effusion.

The **diagnosis** of mediastinal growths is made, if at all, chiefly by exclusion, but it is manifestly impossible before the development of the tumor has progressed to considerable dimensions.

*Aneurysm* is differentiated from solid mediastinal tumors with only slight success in many instances. It is most valuable to note carefully the length of time the condition has lasted, since aneurysm runs a longer course, on the average, than mediastinal tumor, though to this general rule there are many exceptions. The tumor when due to aneurysm communicates a strong, heaving, expansile pulsation—a characteristic that is absent or only feebly manifested in the case of solid mediastinal growths. The severe diastolic shock, as noted both on palpation and auscultation in cases of aneurysm, is also absent in solid tumor. The bruit in aneurysm has often a booming quality that does not belong to the bruit of solid growths. Pain is more pronounced in aneurysm.

The duration of the disease is rarely less than six, and quite as rarely it is more than eighteen, months.

The **prognosis** is absolutely hopeless, except in the case of benign tumors, which may be removed in some instances.

The **treatment** is directed toward the relief of the most urgent symptoms. Anodynes are required sooner or later, and should not be withheld if indicated. As a routine the preparations of iodine and mercury are employed, but, as these are useless, they are unwarranted. Arsenic has sometimes seemed to influence sarcomatous and lymphadenomatous growths favorably, though only temporarily.

(c) **Diseases of the Thymus Gland.**—Nothing is known definitely concerning the functions of the thymus gland, and the diseases of this organ are without special clinical significance. Tumors may have their origin in the thymus gland, and the organ may become enlarged on account of the presence of true hypertrophy or abscess; these conditions are indistinguishable from mediastinal tumor or abscess as above described. Persons who manifest the hemorrhagic diathesis or those who suffer from hemorrhagic affections may also show hemorrhage into the thymus gland—a condition that is identical with that produced by hemorrhage into the mediastinum.

(d) **Mediastinal Hemorrhage.**—This term signifies hemorrhage into the mediastinal connective tissue. It oftenest results from the rupture of aneurysms of the arch or of the large vessels within the thorax, or it may be of traumatic origin (wounds, fractures).

PART V.

DISEASES OF THE CIRCULATORY  
SYSTEM.

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I. DISEASES OF THE PERICARDIUM.

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PERICARDITIS.

**Definition.**—An inflammation of the serous covering of the heart.

**Varieties.**—(a) Plastic, or fibrinous; (b) sero-fibrinous, or subacute; (c) purulent; (d) hemorrhagic; (e) adhesive. There is also a tuberculous pericarditis which has been described (*vide* Tuberculosis, page 308).

ACUTE PLASTIC OR FIBRINOUS PERICARDITIS.

**Pathology.**—The morbid changes are frequently localized, and less frequently are general. At the onset the membrane is smooth, swollen, and injected, and punctured ecchymotic spots may be visible; soon it presents a grayish, roughened appearance in consequence of a deposition of a thin layer of fibrin. In the severer types the fibrinous deposit increases in thickness for a time, and the natural movements of the pericardial surfaces upon one another sometimes cause the exudate to assume a honeycombed appearance. Most examples that I have seen, however, have resembled the roughened surfaces produced by separating two slices of bread that had been thickly buttered; the surfaces are grayish-yellow in color. In the later stages the exudation becomes partly organized, and as the result of friction produced between the opposed surfaces by the incessant action of the heart, the pericardial surface may present a villous appearance; hence the term “hairy heart” which was employed by ancient authors. For like reasons we may see the exudate arranged in the form of little ridges, forming a “tripe-like membrane.” Though invariably present, the amount of serous effusion, as the term would indicate, is never large in dry or plastic pericarditis. Myocarditis may frequently be found as an associated condition.

**Etiology.**—In each variety of pericarditis there are factors that cause the particular form with such relative frequency as to make it desirable to give its etiology separately, except in the sero-fibrinous and acute plastic types, which have practically the same etiology. The two latter are the more common forms of the disease. Acute plastic pericarditis frequently occurs in the young and middle-aged, and is only

rarely a primary process, being secondary to acute articular rheumatism (in more than one-half the cases), to chronic nephritis, and, rarely, to the acute infectious diseases. It may be caused by direct extension of inflammation from adjacent structures, and in this manner it may be a sequel of simple pleurisy; more frequently the extension occurs from a pneumonia or tuberculous pleurisy, or the condition may complicate new growths and inflammatory conditions affecting the esophagus and bronchial glands. It may also be secondary to chronic disease of the aortic valve, the pericardium becoming involved by extension through the walls of the aorta. Finally, it may be the result of traumatism, and in this connection it should be pointed out that this factor may under certain conditions cause any of the other forms of pericarditis.

**Bacteriology.**—Rudini's experiments have shown that the staphylococcus aureus may be a cause of pericarditis, but they have not conclusively demonstrated that it is the specific cause, as is evidenced by the fact that the disease is sometimes caused by other organisms and found in diseases in which other organisms are active. Moreover, staphylococci have not been encountered without demonstrable cause, so that a distinctively specific cause has, as yet, scarcely been proved. Among other organisms, pneumococci, streptococci, and the bacillus coli may be named.

**Clinical History.**—Owing to the fact that acute plastic pericarditis is usually a secondary affection, the symptoms that enable one to recognize it are obscured by the disease of which it is a sequel. This is particularly true of that large class of cases that develop in acute articular rheumatism, in which subjective symptoms are often entirely wanting. It is only in the severest types of this sort that the symptoms referable to the heart are well enough marked to arrest the attention. There may be a feeling of distress or constriction with or without slight pain in the pericardium. During the first stage or prior to the pouring out of the effusion the pain is most marked, extending sometimes into the left arm or the back, and at others to the ensiform cartilage or even to the abdomen. This pain is, rarely, increased by pressure over the pericardium. Palpitation and dyspnea may be present, and the pulse is increased in frequency and strength, as a rule, except in the later period, when it may be weak and slightly irregular, particularly if the muscular tissue of the heart be involved. There is some fever, but the degree of elevation of temperature perhaps never exceeds 102° F. (38.8° C.). In this class of cases the urinary features depend largely upon the character of the leading etiologic factors, though in many instances the urine is scanty, high-colored, and acid in reaction.

**Physical Signs.**—*Inspection* discloses an increased vigor of the apex-beat. Friction fremitus (due to the rubbing of the altered pericardial layers upon one another) may sometimes be felt during the earlier and later courses of the disease or when the membrane is comparatively dry, and is usually most intense near the base, just to the left of the sternum. *Percussion* gives negative results. *Auscultation* usually reveals a double friction-sound over a limited area in the precordia—a characteristic sign, though one on which sole reliance must not be placed in this disease. The friction-rub is caused partly by the exudate and partly by the dry state of the membrane. Its usual seat of maximum pronunciation is in



the fourth and fifth interspaces and the adjacent portions of the sternum—*i. e.* that portion of the heart which is most closely in contact with the front of the chest (Osler). Another favorite point is the cardio-aortic junction. It is usual to hear the rub over small areas, though occasionally it is audible over the whole precordia, and its distinguishing feature is its superficiality, being generated close to the ear. Pressure with the stethoscope, which approximates the layers, increases its intensity, though if too much force be exerted the murmur may disappear entirely. In like manner the friction-sound is influenced by respiration and change of posture. The quality of the sounds, like their position, exhibits great variability. They are sometimes soft, but most frequently are grating or rubbing, and in the later stages I have noticed that they may have a loud creaking quality, closely simulating the bending of new leather. Though with few exceptions they are double, and are primarily produced by the rhythmic movements of the heart, they do not always occur synchronously with the heart-sounds, and usually exceed the latter in duration—facts that go to show that the quality, location, or superficial area of a given murmur in no wise indicates the extent of the pathologic process.

**Complications.**—There may be an extension of the inflammatory process to the external surface of the pericardium, either from the deeper pericardial structures or from the pleura, particularly the left. This is a complicated condition termed "*external pleural pericarditis*" or "*mediastino-pericarditis*," in which the mediastinal connective tissue is also, as a rule, involved. It is most frequently secondary to tuberculous pleurisy (*tuberculo-mediastino-pericarditis*), sometimes also to pleuro-pneumonia, and rarely to simple pleurisy or plastic pericarditis. The recognition of these combined lesions rests chiefly upon the detection of a friction-murmur that is partly dependent upon the cardiac and partly upon the respiratory movements. These sounds are most distinctly heard along the left edge of the heart. Momentary arrest of breathing suppresses the pleuritic friction-sound, there remaining merely the sounds produced by the rhythmic cardiac action, and even these may be absent. On the other hand, during forced respiration nothing is audible, as a rule, except the strong pleural rub. In normal respiration the inspiratory movements decrease while expiratory movements increase the intensity of the sounds. During inspiration the *pulse* may become small and slow, owing to the partial occlusion of the aorta, brought about by the traction of fibrous bands of adhesions which pass over the vessel, being at the same time connected with the pleura. When these bands pass from the exterior of the heart-muscle or pleura, they may cause, as first pointed out by Riegel, an absence of the apex-beat during expiration. Instances of this sort are not uncommon.

**Diagnosis.**—Although the presence of a to-and-fro friction-sound is, as a rule, indicative of plastic pericarditis, it is an error to regard it as an infallible sign, since complete calcification of the coronary arteries, as well as excessive dryness of the pericardial surfaces, may rarely produce friction-murmurs.

**Differential Diagnosis.**—The harsh double murmurs due to *chronic valvular lesions* can be eliminated if it be recollected that they are more constant, more distinct, and that each has its area of transmission beyond

the limits of the precordia. The sitting posture, leaning forward, or moderate pressure with the stethoscope, all fail to produce or to increase *endocardial murmurs*, whether acute or chronic. A double aortic murmur is associated with cardiac hypertrophy, the Corrigan pulse, and systolic flushing of the capillaries.

**Prognosis.**—The termination is always favorable as to life. Complete resolution does not often occur, but the exudate becomes connective tissue, and agglutinates the two layers of the pericardial sac. The acute may merge into the chronic form, and dry, plastic pericarditis often constitutes the first stage of severer grades of the disease.

**Treatment.**—Absolute quiet in the recumbent position should be enjoined. The diet should be composed chiefly of light, easily digested solids, allowing as little drink as is practical, and thus endeavoring to avoid an overfilling of the vessels. With the same object in view, if the patient's strength be good, a half-dozen leeches should be applied over the heart, followed by the use of the ice-bag; the bowels are to be kept soluble by using stewed fruits or saline laxatives if needful. Calomel in doses ranging from gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  (0.016–0.032) every hour or two, combined with a little opium to prevent purgation, is serviceable. At the beginning veratrum viride may also be cautiously administered, with a view to dilating the arterioles throughout the rest of the body, and thus virtually "bleeding the patient into his own vessels." Later, digitalis in combination with the iodids of potassium and iron should be substituted for the purpose of absorbing the effused material. Tonics and a change of air may be required during convalescence.

#### SERO-FIBRINOUS PERICARDITIS.

**Pathology.**—The anatomic changes may be grouped into three stages—the *first* being characterized by a plastic exudation (corresponding with the lesions in dry, plastic pericarditis, though more pronounced); the *second stage*, by a variable amount of effusion composed largely of serum. The exudation usually begins about the origin of the great vessels springing from the base of the heart, and ultimately forms a thick covering of fibrin, especially on the visceral layer. The quantity of serous effusion may be from 2 to 10 ounces (64.0–320.0), but occasionally it is as much as 3 pints ( $1\frac{1}{2}$  liters). The admixture of a slight amount of blood- or pus-corpuscles sometimes occurs in this form of the complaint. The *third* is the stage of absorption in the most favorable cases. Perfect resolution rarely takes place, but, instead, the liquid effusion is alone absorbed, and the lymph causes firm adhesions of the visceral and parietal membranes. If, as sometimes happens, the serum remains, the acute passes into a chronic condition. The muscular tissue of the heart may become involved by an extension of inflammation from the visceral layer which lies in contact with it; it is always the seat of more or less collateral edema. The grade of the myocardial inflammation will depend much upon the extent and duration of the pericarditis, though usually it is moderate in the fibrino-serous variety.

**Etiology.**—The disease is most frequently observed to be associated with acute rheumatism, Bright's disease, and pulmonary tuberculosis. Respecting the causal relation of acute rheumatism, two facts should be



stated—viz. that pericarditis arises as a complication in about one-third of the cases (Bamberger), and that it may rarely precede, by a few days, the articular disturbance. I am of opinion that in exceptional instances both sero-fibrinous and plastic pericarditis may occur secondarily to the rheumatic dyscrasia without the slightest evidence of arthritis. The disease also occurs in the course of the eruptive fevers, and sometimes as a complicating affection due to extension of inflammation from neighboring parts.

**Clinical History.**—When, as rarely occurs, a *primary* pericarditis develops, the initial symptoms common to inflammation of other serous membranes manifest themselves, as anorexia, sometimes nausea and vomiting, chills, fever, increased respiration and pulse-rate, together with local pain. The pain is usually of a dull, aching character, and less frequently merely a slight soreness, or it may be absent altogether. Acute pain is experienced only when the pleura is implicated.

When pericarditis is *secondary* to an existing febrile affection there are, in many cases, no subjective symptoms to indicate its presence. In other instances there may be precordial oppression with or without slight pain or a feeling of soreness. Hence the rule should be absolute that in all affections in which pericarditis is likely to arise physical examinations of the heart should be frequently made, and particularly during the height of the disease.

Dyspnea comes on simultaneously with the appearance of the effusion and may lead to actual orthopnea. Pressure is exerted upon the left lung if the effusion be large—a fact that explains in part the presence of dyspnea. The cardiac muscle, especially the right ventricle, is also pressed upon by the effusion, thus impeding to a greater or lesser extent the cardio-pulmonary circulation as well as the cardiac diastole. We have here an additional reason why dyspnea occurs, and also why deficient aëration of the blood and a feeble peripheral circulation are found in this complaint. Prior to the occurrence of the effusion the circulation is too actively carried on, the pulse being full and strong. It is clear from the above explanation relative to the mechanical effects of large effusions that during the second stage the pulse is small, feeble, and irregular. When the liquid effusion is not large the heart-action may be apparently feeble, while the pulse remains strong—a valuable rational sign. On the other hand, an excessive amount of fluid may cause the radial pulse to become quite small or even to disappear during inspiration (the *pulsus paradoxus*). Fever is present, as a rule; the temperature is irregularly elevated, ranging from 101° to 103° F. (38.3°–39.4° C.). In favorable cases defervescence takes place by lysis. Nervous symptoms, as headache and mild delirium, often appear, and sometimes give place to stupor or even coma.

**Physical Signs.**—*Inspection.*—The skin-surface and mucous membranes are observed to be pale and more or less cyanotic. The neck-veins are prominent, and sometimes exhibit undulatory movements or pulsations. The face wears an anxious expression; the respirations are increased, labored, and at times irregular. The decubitus is dorsal; the head and shoulders are elevated, and the patient may be forced to assume the sitting posture. In young subjects precordial prominence, with effacement or even bulging of the intercostal spaces, may result from the pres-



ence of a moderate effusion. In adults, however, a large collection is indispensable for the production of this effect. If the lung be shrunk or if there are pleuritic adhesions, expansion of the pericardium, and hence also bulging, will be prevented. The distended pericardium may depress the diaphragm. Elevation of the left nipple in consequence of marked anterior expansive bulging has been observed. In the first stage the apical beat is intensified, but as the effusion increases (forcing the heart backward and upward) it is displaced in an upward and outward direction, at the same time becoming weaker as well as more diffused, since with expansion of the sac comes greater mobility of the organ. When the pericardial sac becomes filled the impulse-beat disappears, for the reason that the fluid now completely surrounds the heart and pushes it away from the chest-wall.

*Palpation* confirms the result of inspection. The apical beat is diffused and feeble or lost. When detectable it is found to be displaced upward and to the left. Altering the patient's posture changes the seat of the apex-beat (Oppolzer), and if the shock has been lost, turning the patient on his left side or bending his body forward may cause its return. The cardiac impulse disappears earlier when, on account of myocarditis, the systole is greatly enfeebled. On the other hand, old adhesions may retain the apex-beat in contact with the chest-wall, despite the presence of a large accumulation. Hypertrophy of the organ would act in a similar manner, though less potently. A friction-rub can be felt occasionally over the base of the heart even when there is a copious effusion present, and, if absorption takes place, the friction fremitus becomes more marked, simulating the first or dry stage.

*Percussion.*—The area of cardiac dulness is greatly increased, and assumes a characteristic triangular form with the base downward and the apex extending up to the third or even second interspace to the left of, though near, the sternum. The lateral border-lines of dulness obviously diverge from above downward, the right passing to a point corresponding with the right edge of the sternum, along which it runs to the seventh rib; the other to the left, finally intersecting the base-line at the left anterior axillary line. The lower level of the fluid, being continuous with the liver dulness, is not definable. Even in moderate effusions there is flatness in the fifth interspace to the right of the sternum (Rotch). The margins of the lungs surrounding the heart may be retracted and the heart carried forward or dilated, owing to the presence of adhesions; the dull space will then appear larger than is justified by the amount of fluid. Retraction or moderate compression of the lung may, however, give rise to a modified tympanitic resonance to the left of the flat area. Occasionally the lung is attached anteriorly, and then the heart is crowded backward by the effusion, while the area of flatness on percussion is relatively diminished. The triangular shape of the flat space, noted when the patient is in the sitting posture, is to a considerable extent lost and its area diminished when he changes to the supine position or lies on either side, the effusion obeying the laws of gravitation. When the feeble impulse can be felt by the clinician occupying the center only of a dull area, he has good evidence of the existence of pericardial effusion.

*Auscultation.*—The characteristic friction-rub of the first stage has

already been described. It may, however, also be audible over the base during the stage of effusion, and always returns, after absorption of the fluid, for a brief period. The heart-sounds grow more and more distant, faint, and muffled, though the second sound, as heard over the extreme base of the organ, may remain clear. Over the area of dull tympany corresponding to the lower antero-lateral portion of the left lung (which, as before pointed out, is more or less compressed) may be heard bronchovesicular breathing.

**Course and Duration.**—It will appear obvious that the course must vary in individual cases with the cause and severity of the special type of infection. Observation has shown that in one class of cases the three stages (dry, effusion, and absorption) are passed through in rapid succession, while in another class each stage is proportionately lengthened. The latter type has been termed “chronic” by some and “subacute” by others. The acute may also be followed by the chronic variety. Usually sero-fibrinous effusions complicating rheumatism are absorbed with rapidity once the process has begun, seldom requiring more than two weeks. When recovery is about to occur the temperature falls by lysis; the dyspnea gradually disappears, and with it the effusion is gradually absorbed. Convalescence is further indicated by a return of the appetite, normal heat of the skin, and a less frequent, full, and regular pulse. In cases that tend to a fatal termination either the fever continues or there is suddenly developed hyperpyrexia, as may happen when pericarditis occurs in the course of acute rheumatism; in such cases the dyspnea is urgent and cyanosis is often marked, with signs of failing circulation. *Nervous symptoms*, as extreme restlessness, insomnia, and active delirium, may be present. Under these circumstances death usually ensues at the end of a week or ten days. In a fatal case of *acute articular rheumatism* which I saw, complicated by pericarditis, with hyperpyrexia, death occurred on the sixth day. Rarely *acute pleuritis* with effusion is a complication, and its occurrence usually lengthens the course of the pericarditis and renders the outcome of the latter condition uncertain. When there coexists *extensive myocarditis*, syncopal attacks often endanger the life of the patient. Copious effusion may, by causing pressure upon the recurrent laryngeal nerve, produce paralysis of the vocal apparatus; or, as the result of pressure upon the esophagus, difficult deglutition may be a troublesome concomitant.

**Prognosis.**—In sero-fibrinous pericarditis recovery is the rule under favorable conditions. The outlook, however, becomes gloomy when the above-mentioned complications arise, and particularly when there is hyperpyrexia in connection with acute rheumatism. Occurring as a secondary event in serious acute diseases, as pneumonia, or in chronic diseases, as Bright's, or organic affections of the heart, the pericarditis often precipitates a fatal termination. The strong possibility that these cases may only partially recover or assume a chronic form must be recollected in making a prognosis.

**Diagnosis.**—The disease is often overlooked, because unsuspected. Ordinarily the recognition of pericarditis by the characteristic triangular area of percussion-dulness and by the friction-sound is not difficult. Atypical cases or those first seen during the stage of effusion can only be correctly diagnosticated by exclusion.



**Differential Diagnosis.**—*Acute pleurisy* of the left side may simulate pericarditis with copious effusion, and, as before stated, these diseases may coexist. Acute pain, however, belongs to pleurisy alone. In pericarditis the characteristic physical signs are elicited over the precordia; in pleurisy they are apt to occupy not only the anterior but also the axillary and posterior aspects of the chest; hence the percussion-flatness in pleurisy extends to the left, far beyond the boundary-line of the percussion-flatness in pericarditis. The pericardiac friction-sound has a different situation usually from the pleuritic, and the latter is heard synchronously with the respiratory movements, while the former is intimately related to the time of the cardiac movements. The friction-murmur of pleurisy ceases if the breathing be momentarily suspended. Encapsulated pleural effusions that are limited in area to the antero-lateral portion of the chest are exceedingly difficult of elimination, and especially in the absence of pleuritic friction. In the latter complaint, however, the heart-sounds are clear and the apex-beat often pushed some little distance to the right; on the other hand, in pericarditis the general disturbance is usually greater, while a friction-rub may be detectable over the base. The heart-sounds are distant and muffled. The diagnosis is often aided by a consideration of the previous history and the bearing of any facts thus obtained upon the known etiology of these affections. We encounter intricacies when we attempt to exclude *cardiac dilatation*, though the following brief table will be of assistance in the diagnosis:

PERICARDITIS WITH EFFUSION.	CARDIAC DILATATION.
( <i>Previous History.</i> )	
Recent history of gout, acute rheumatism, acute infectious or septic disease, scurvy, chronic nephritis, or tuberculosis.	Usual history of chronic and valvular disease of the heart.
( <i>Clinical History.</i> )	
Fever and slight pain are usually associated.	No fever or pain, as a rule.
Nervous symptoms are often present.	Absent.
( <i>Physical Signs.</i> )	
<i>Inspection</i> often reveals bulging (more marked in the young). Apex-beat pushed up, is feeble, and later absent. Heart's impulse usually absent. Friction-fremitus may be present over the base.	Apex-beat usually visible, wavy, and diffused.
<i>Percussion</i> shows a triangular flat area, and the boundary-line above changes on altering the position. There is dull tympany in the axillary or subscapular region.	Though feeble, the impulse is palpable.
<i>Auscultation</i> shows the first sound distant and muffled; the friction-rub is often double over the base.	Dull area varies with chambers dilated: usually it is coextensive with a wavy impulse, and does not extend so high (except in mitral stenosis), and does not vary with change of position. No dull tympany.
	First sound clear, short, and sharp. No friction-murmur present, but an endocardial murmur or murmurs may appear.

**Treatment.**—The management of the first (or dry) stage is identical with that detailed in discussing the plastic variety. During the stage of effusion the patient should be kept at absolute rest in the recumbent posture, and mental excitants should be rigidly prohibited with a view to



minimizing the labor of the heart. The diet is to consist mainly of easily digested albuminous articles; fluids are not to be given in large amounts, since this tends to overfilling of the vessels, increases the arterial tension, and delays absorption.

*Local Measures.*—Flannel should be kept over the precordia, so as to avoid exposure and undue chilling. The ice-bag or Leiter's coils (to be used in the first stage) should be cautiously employed during the second stage, until the temperature has defervesced considerably, thus indicating a subsidence of inflammation in the pericardium.<sup>1</sup> Subsequently, if absorption does not proceed satisfactorily, blisters may be applied over the precordia, and, should the patient's general condition be markedly asthenic, an ointment containing iodine, lanolin, and ichthyol may be substituted with advantage. The *therapeutic measures* must be chosen with sole reference to the primary disease, which the physician must continue to treat while he attempts by other means to relieve certain symptoms and promote absorption. For example, if the pericarditis be due to rheumatism, the use of the salicylates must be persevered in, and opium may be added to quiet restlessness and procure relief from pain. In my own experience absorption has been best promoted by the use of the double iodide of potassium and iron or of iron and manganese. These agents are seldom contraindicated unless they are badly borne by the stomach. Diuretics and saline purgatives are not without value, but do good only in the later stages. Depressing measures of whatever sort are not to be resorted to unless the circulation be good. If the pulse be small, weak, and rapid, with marked cyanosis, stimulants are indicated and are to be given in moderate quantity; the pulse will then be found to grow stronger and the dyspnea and cyanosis less marked. The nervous symptoms are also benefited as a result of the action of these agents. Strychnin and the salts of ammonium will be found to be useful. Digitalis and strophanthus are not to be thought of when myocarditis is associated; at other times they often improve the peripheral circulation and increase the urinary secretion. When the breathing becomes greatly embarrassed and the circulation fails, as shown by the feeble, broken, rapid pulse and the cyanotic hue of the lips, eyelids, and finger-tips, cardiocentesis is indicated, and in sero-fibrinous effusion aspiration has, in recent years, given good results if not too long delayed. If the slightest doubt arises as to the character of the fluid, a preliminary puncture with a hypodermic needle should be made. The point for puncturing is the fourth interspace, 1 inch (2.5 cm.) from the parasternal line, or the fifth interspace, 1½ inches (3.7 cm.) from the left edge of the sternum. The operation must be performed with the strictest asepsis, and the amount of liquid withdrawn at any one time should not exceed two or three ounces. It would be better to repeat the puncture several times than to remove the pressure too suddenly from the damaged heart.

#### PURULENT PERICARDITIS.

(*Empyema of the Pericardium.*)

**Pathology and Etiology.**—The condition often follows the sero-fibrinous form. Septic and tuberculous processes involving the pericar-

<sup>1</sup> If the pericarditis be secondary to an acute febrile disease, this fact must modify the method here recommended accordingly.

dium are also apt to cause purulent effusion, and many of the cases that arise in the course of the acute infectious diseases belong to this category. The membrane is much thickened and presents a gray, granular surface, and the myocardium underlying the visceral layer is softened, fragile, and pale-looking—changes that are in the main due to fatty degeneration.

**Clinical History.**—The local subjective symptoms and physical signs are the same in kind as in the former variety, but the amount of exudation is frequently less. At the onset rigors often occur, and may be repeated at varying intervals. The temperature-curve is of the suppurative type; the pulse is small, rapid, and irregular; and physical prostration is pronounced. Purulent pericarditis runs a comparatively rapid and an almost uniformly unfavorable course.

**Diagnosis.**—The chief clinical features are often referable to the primary or causal disease; hence in every instance in which purulent pericarditis is apt to arise a physical exploration of the chest is imperative. The purulent character of the effusion cannot readily be ascertained, as a rule; but the history of an affection having etiologic importance, the observance of rigors, and the presence of the fever-curve peculiar to suppuration would all point strongly to purulent effusion, and should lead to aspiration with the hypodermic needle—a harmless procedure if carefully performed, and one that almost constantly gives reliable results.

**Treatment.**—It is within the physician's province to treat the primary disease assiduously, but he should not undertake to treat complicating pericardial empyema by the application of therapeutic measures. A surgeon's aid should be invoked.

#### HEMORRHAGIC PERICARDITIS.

In purulent pericarditis the effusion may be hemorrhagic, and particularly when it is of tuberculous origin. In non-purulent tuberculous pericarditis also the exudation is apt to be hemorrhagic. In the non-purulent instances that are due to chronic Bright's disease or that occur in the aged the effusion is sometimes blood-stained; and future observation may show that the hemorrhagic variety is of more frequent occurrence than has hitherto been supposed. Here may be pointed out that even in ordinary serous pericarditis there is apt to be much more blood than in serous pleuritis. M. T. Ferrier has found 5 examples in 9 collections. This etiologic variety scarcely calls for separate clinical consideration.

#### ADHESIVE PERICARDITIS.

(*Chronic Pericarditis.*)

**Pathology and Etiology.**—Chronic pericarditis follows the acute forms, and, as in the case of the latter, it may be partial or general. The effusion may rarely remain as a permanent condition, though not infrequently a clear history of the preceding acute attack is wanting. In most instances the opposed surfaces of the membrane are either universally or over a limited area firmly adherent. The amount of new connective tissue present or the degree of thickening of the layers varies greatly, and is dependent upon the type of the primary acute attack. If



the latter is of mild grade—as, for example, in the case of the sero-fibrinous variety, complicating rheumatism—then not much thickening is encountered in the resulting chronic form.

*Chronic tuberculous pericarditis* is not uncommon, and may be primary, though more commonly it is secondary, in its origin. The disease is not invariably preceded by the acute form, but may be chronic from the time of onset. I have noticed that often more or less effusion prevails unless artificially removed. The layers become enormously thickened, and total obliteration of the sac by agglutination of the surfaces is not infrequent.

In the dense exudate that remains after complete absorption of a pericardial effusion calcareous depositions occur, forming a bony casing, as it were, which either partially or totally encircles the organ. The external surface of the pericardium may become united either with the costal or pulmonary pleura, the chest-wall, or the mediastinal tissues. The myocardium is the seat of atrophic and degenerative changes.

**Symptoms.**—Autopsies frequently reveal a chronic adhesive pericarditis that has not given rise to a single recognizable symptom during life. Hypertrophic dilatation of the chambers usually develops sooner or later, and is due to adhesions that interfere with the free action of the organ as well as with its systole. When present the subjective symptoms point to enfeeblement of the cardiac muscle, as shown by the universal venous stasis. The symptoms of bronchial and gastro-intestinal catarrh are often prominent; rational symptoms indicative of adhesion are, however, scanty and unreliable. The pulse is rapid, of low tension, and irregular, and, though not diagnostic, the *pulsus paradoxus* has been noted.

**Physical Signs.**—*Inspection.*—Depression or pitting of the intercostal spaces over the position of the heart may be noticed. Synchronous with the systole there is also a retraction of the chest-wall in the apical area, and less frequently over the whole precordia, the latter being an unerring sign of universal adhesions. The degree of systolic retraction is slightly influenced by the respiration, inspiration increasing it, and it is best appreciated on palpation while the patient is in the semi-supine position. During the diastole the heart forcibly rebounds, causing the so-called diastolic shock, which is of the utmost diagnostic worth when associated with marked systolic retraction. Though not always visible, it can be readily felt on palpation. Friedrich's sign (the sudden collapse of the jugulars during diastole) may frequently be observed, but I have also noticed this in cardiac dilatation without adhesions. Prior to the onset of dilatation the apex-beat may be forcible and visible over an increased area, indicating hypertrophy; but after the myocardium is weakened (from interference with its nutrition) and dilatation comes on, the impulse-beat is faint or wanting, and in marked systolic retraction may be seen to be vibratory. The fixed position of the apex-beat when the patient is turned over upon his left side is a confirmatory sign of considerable value.

*Percussion.*—The area of cardiac dulness is increased, especially upward and to the left, owing to the associated hypertrophy and pleural pericardial adhesions, and, since the adhesions between the pleura and the pericardium do not allow the lungs to come forward and overlap the heart



during inspiration, the upper and left lines of dulness remain fixed (C. J. B. Williams).

**Auscultation.**—When dilatation reaches a high degree the auscultatory signs peculiar to that condition appear. In many cases no murmurs are detectable, but in a third group loud murmurs, quite independent of any value as regards cardiac lesions, are audible; these murmurs may be due to the vortiginous movements in the antero-cardial blood-current occasioned by the jogging cardiac action. Finally, it is to be noted especially that chronic adhesive pericarditis may exist without giving rise to any physical signs.

**Differential Diagnosis.**—The condition is apt to be confounded with *chronic myocarditis* and *simple hypertrophic dilatation*.

As before stated, chronic pericarditis may be associated with effusion, and in such instances it is important to distinguish from the adhesive type if we would institute proper treatment. In chronic pericarditis with moderate effusion the seat of the apex-beat is higher and less undulatory, and when the amount of effusion is large the impulse is absent and there is bulging. Adhesive pericarditis with hypertrophy causes bulging in young subjects, but the apical beat is retained. In pericarditis with effusion the upper and left limits of dulness are not stationary, and there is an absence of systolic retraction and diastolic concussion.

**Course and Prognosis.**—The hypertrophy that comes on early in consequence of the obstruction offered to cardiac action is compensatory, and this harmonious balance may be maintained for a long period of time with apparent comfort. After myocardial degeneration, followed by atrophy or dilatation, has occurred, the condition becomes quite serious, and death usually ensues amid signs of extreme cardiac dilatation.

The **treatment** must be ordered chiefly with reference to the nutrition of the heart-muscle, following the principles noted in dealing with the management of valvular affections of the heart. If chronic effusion be present early, operative measures are to be warmly advocated.

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## HYDROPERICARDIUM.

(*Dropsy of the Pericardium.*)

**Definition.**—A condition in which the pericardium contains a serous transudation, while the membrane itself shows no signs of inflammation.

**Etiology.**—(a) Hydropericardium is usually associated with general cardiac or renal dropsy, of which it forms a component part. Under these circumstances it develops late, and frequently follows hydrothorax, on account of which condition it is liable to be overlooked. It may also occur suddenly in chronic nephritis, and particularly in the scarlatinal variety. (b) It arises not infrequently from local mechanical causes, as the pressure of mediastinal tumors, aneurysm, or thrombosis of the cardiac veins.

**Symptoms.**—No subjective symptoms are present, save perhaps dyspnea, and the diagnosis rests upon the history and the physical signs. None of the latter, however, are particularly significant. They point to

the presence of fluid, and the area of percussion-dulness assumes the same form and exhibits even greater change, with alteration of the patient's posture, than in pericarditis. No friction-murmurs are heard and no bulging of the pericardium is observed. It is rare indeed, I have found, to see an excessive amount of serum in the pericardium at the *postmortem*. The symptoms and signs of hydrothorax generally precede and accompany hydropericardium, and the latter condition tends to intensify the effect of the former. The condition, *per se*, is rarely of serious import. Osler remarks: "Naturally there are in the pericardial sac a few cubic centimeters of clear, citron-colored fluid, which probably represents a post-mortem transudate." In rare instances the transudate has a milky appearance (*chylo-pericardium*).

The **treatment** suitable for cases of general dropsy, as a rule, affords relief. In large serous accumulations aspiration should be practised.

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### HEMOPERICARDIUM.

By the term "hemopericardium" is meant hemorrhage into the pericardial pouch—a rare event. Among the causes are—(a) perforation by aneurysms of the aorta and the coronary arteries into the sac; (b) rupture of the heart, due to injuries or cardiac aneurysms and fibrous formations from myocarditis; (c) direct injuries, especially stab- and bullet-wounds. *The symptoms and course* depend greatly upon the nature of the exciting cause. The most frequent factor, rupture of an aneurysm, proves quickly fatal from overcrowding of the heart. In rupture of the heart-muscle there is sometimes a slow outpouring of blood, with a correspondingly slow course, varying from a few hours to a couple of days in duration. The physical signs of effusion come on with dyspnea and failing circulation, which lead to cardiac exhaustion and death. The blood-stained effusions, before considered, that are met with in certain forms of pericarditis, are not to be regarded as instances of hemopericardium.

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### PNEUMOPERICARDIUM.

(*Air in the Pericardium.*)

In this complaint, besides air or gas, there is usually present pus, and less frequently blood; hence an appropriate term in most instances would be *pyo-pneumopericardium*. When the pericardium is perforated purulent pericarditis results. *The causes* are the following: (a) wounds; (b) a fistulous connection between the adjacent air-containing organs and the pericardium as the result of diseased processes, such as pulmonary tuberculosis or empyema; (c) rarely decomposition of liquid pericardial effusions. *The symptoms* are equivocal. In the main they do not differ from those of pericarditis with effusion, excepting that dyspnea is more intense than in the latter affection. By attention to the physical signs

the distinction from pericarditis can rarely be made. In pneumopericardium there is tympanitic percussion-resonance over the precordia, though the fluid, when present, gives rise to a boundary-line of dulness. The change of the patient's posture alters markedly the area of the tympanitic note. On auscultation may be heard loud, rasping, friction-sounds having a metallic quality, intermingled with churning, splashing noises, or the so-called "water-wheel sounds." I have, however, occasionally found the apex-beat and heart-sounds exceedingly feeble. Pneumothorax when encysted in close proximity to the heart, displacing the latter organ, must be eliminated. The latter complaint gives cardiac dulness in the abnormal position and a metallic sound synchronously with the respiratory movements—two signs diagnostic of pneumothorax that are absent in pneumopericardium. *The prognosis* is grave, death coming on most commonly in a day or two. The admission of air might alone result in a spontaneous cure, as occurs rarely in pneumothorax. *The treatment* is the same as has been recommended for purulent pericarditis.

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## II. DISEASES OF THE HEART.

### ENDOCARDITIS.

**Definition.**—Inflammation of the lining membrane of the heart. The process is usually confined to the valves, though the cardiac layer may also be affected.

**Varieties.**—(a) Simple acute endocarditis; (b) ulcerative endocarditis; (c) chronic endocarditis. The pathologic processes involved in the first two, the acute forms, are identical in nature, though they differ in severity. I have met with two instances that could be referred to neither sub-variety, apparently occupying a middle ground.

#### SIMPLE ACUTE ENDOCARDITIS.

(*Endocarditis Verrucosa.*)

**Pathology.**—The disease is characterized by the formation of small vegetations on the segments, varying in size from excrescences that are scarcely visible to those the size of a pea. They are found chiefly on surfaces that are opposed to the blood-current, near the margin of the valve, and "forming a row of bead-like outgrowths." Their seat corresponds to the point of maximum contact (Sibson), but the mitral valve is much more commonly affected than the aortic. With the segments the chordæ tendineæ are sometimes affected, and very rarely the latter are alone involved. The left side of the heart is much more frequently the seat of acute endocarditis than the right, except during fetal life, when the right side is almost exclusively involved. To account for the greater frequency of occurrence on the left side after birth, it has been suggested that freshly oxygenated blood affords the most favorable condition for the multiplication of the micro-organisms that are concerned in the inflammatory process. As corroborating this view, the fact is adduced that during



fetal life the blood in the right chamber is the more completely oxygenated. It has also been pointed out that before birth the right side, and after birth the left side, is the more active, and that the active side is apt to suffer on account of higher pressure. Obviously, the vegetations form an obstruction to the current of the circulation as it flows through the valvular opening. In the early stage the membrane in the vicinity of these excrescences shows a bright-red color, which has usually disappeared in fatal cases before they come to autopsy. The histologic changes consist in a proliferation of the subendothelial tissue (small-celled infiltration), which forms the principal component part of the vegetation. On this basal mass of granulation tissue there is deposited fibrin from the blood, the latter being separable from the former in acute forms of the complaint. Micro-organisms have repeatedly been found in the fibrinous depositions, but the specific causal irritant has not as yet been discovered. In favorable cases either the vegetation is ultimately absorbed or there remains a small indurated mass. When the vegetations are of considerable size emboli may become detached by the force of the blood-current, and be carried to the vessels of the extremities and to the various viscera, particularly the brain, spleen, and kidneys, giving rise to embolic infarcts. The latter event is frequently observed in cases in which acute endocarditis is engrafted upon chronic valvulitis.

Simple acute endocarditis may end in the more serious or ulcerative variety. Here the cellular proliferation proceeds actively, leading to necrosis of the newly-formed tissue and to the production of an ulcer. Much more commonly, however, does the simple form terminate in chronic (sclerotic) valvulitis with deformity.

**Etiology.**—The most frequent cause of acute endocarditis is *acute articular rheumatism*, which complicates the disease in not less than 40 per cent. of the cases. In young subjects suffering from rheumatism the liability to the complaint is particularly pronounced. The severity or mildness of the rheumatic attack does not, however, influence the appearance of the cardiac complication. Cases of acute endocarditis of rheumatic origin are met with in which the arthritic phenomena are secondary. It may complicate *tonsillitis* when the latter is due to or associated with rheumatism. In *specific fevers* it is also encountered, and found to be common in scarlet fever, but rare in typhoid fever, diphtheria, measles, erysipelas, variola, and varicella. It is not uncommon as a complication in *pneumonia* and *pulmonary tuberculosis*, and Osler, as the result of 100 autopsies in cases of pneumonia, found it present in 5 instances, while in 216 *postmortems* upon phthisical cases it was present in 12 instances.<sup>1</sup> It has frequently developed in the more serious forms of *chorea*, and intercurrent acute endocarditis may result from chronic diseases attended with emaciation and general weakness or suppuration, such as ulcerative carcinoma, gleet, gout, chronic Bright's disease, and diabetes. Lastly, acute endocarditis may occur as a secondary event in pre-existing sclerotic endocarditis, when it is termed *acute recurrent endocarditis*.

In chronic endocarditis the liability to the acute form is greatly increased by the puerperal state, and also, though to a lesser extent, by pregnancy. It is highly probable that the micro-organisms of inflammation, assisted by the friction between the blood-current and the surfaces

<sup>1</sup> *Text-book of Medicine*, Osler, pp. 628, 629.

of the valves, fix themselves upon the latter and there set up the characteristic lesions. Indeed, the disease has of late been excited by injecting into the blood the streptococcus pyogenes, staphylococcus aureus, and other micrococci. Fränkel and Sängner are of the opinion that the staphylococcus pyogenes aureus is the chief specific agent in causing acute endocarditis, but their claim has not as yet been satisfactorily established.

**Clinical History.**—It is only occasionally that definite subjective symptoms, as precordial pain (sometimes extending down the left arm), dyspnea, and cardiac palpitation, are complained of by the patient. In the vast majority of instances the condition is discovered accidentally in the course of an examination of the chest. This being true, not only its frequent occurrence in acute articular rheumatism, but also the possibility of its occurrence in the other diseases mentioned under “Etiology,” should ever be kept in remembrance. The symptoms of embolism, which will be detailed presently, are rarely observed in this form of endocarditis.

The **physical signs** by which acute endocarditis is recognizable are dependent upon the valvular insufficiencies caused by the morbid lesions previously described. Hence there must be not a small proportion of mild cases, including those in which the valves are not affected, that give rise to no physical signs.

On *inspection* the area of visible impulse may be seen to be increased, though, as a rule, it is normal. The impulse is sometimes forcible and often irregular during the initial period, but later it becomes less distinct and more feeble. *Palpation* confirms the result of inspection. I have found the impulse to vary at each visit, with a general tendency to lessen in intensity in the later period of the disease. A very weak impulse is indicative of associated myocarditis or of the poisonous effect of a severe type of primary infection. In recurrent endocarditis the apical impulse is often heaving, on account of pre-existing compensatory hypertrophy, and its area is exceedingly variable. A systolic thrill is sometimes felt.

On *percussion* the area of the heart's dulness is found to be almost uniformly normal, except in cases of intense myocardial involvement, when acute dilatation of the chambers may supervene, giving rise to an increased area of percussion-dulness in the transverse direction. In recurrent acute endocarditis the area of dulness corresponds to the increased area of the apical beat.

**Auscultation.**—Acute endocarditis is usually attended with a soft blowing, systolic murmur, which, owing to the fact that the mitral segments are the favored seat of the disease, is heard much more frequently at the apex than at the base. The point of maximum intensity of this murmur is often movable, but its area of transmission is usually quite limited. In rheumatic endocarditis this murmur is preceded by a prolongation of the first sound and the consequent shortening of the interval between the first and second sounds. The murmur is sometimes heralded by a feeble or muffled first sound, with intensification of the second, and sometimes by a pronounced accentuation or roughening of the normal sounds. In acute endocarditis affecting the mitral valves aortic murmurs may coexist, but their true nature is more than doubtful. There is also a short, low-toned, and double systolic murmur over the tricuspid orifice in a small proportion of the cases; this is due most probably to a relative incompetency. When acute endocarditis arises in connection with chronic



valvular disease, the auscultatory signs of the latter are but little changed, and hence a positive diagnosis is not possible.

**Complications.**—There may be developed by direct extension secondary myocarditis, a disease that will receive separate consideration.

The **diagnosis** is based on the physical signs, though it must be remembered that these are untrustworthy. The points gained by careful inspection and palpation are as significant as those learned by auscultation, as is also the previous history of the patient. The soft bellows murmur is often present in *acute febrile diseases* in which the autopsy fails to reveal the lesions of acute endocarditis. The functional murmurs that arise in the specific fevers, however, are perhaps oftener heard over the aortic area, while those occurring in endocarditis are heard best over the mitral area. Leube<sup>1</sup> points out that if the dulness is slightly increased to the left, there is fever; in fact, if there is infectious disease present, a diagnosis must be made of acute insufficiency of the ostium mitralis occurring in the course of acute endocarditis. The distinction between simple acute endocarditis and pericarditis should be categorical, in view of the manifold differences between their signs. But the fact that these two affections may be associated, more especially when they are of rheumatic origin, must be steadily borne in mind, and also that when combined the signs belonging to the endocarditis are not open to observation, owing to the pericardial friction-sound, and later the presence of the effusion. I have found, however, that, fortunately, endocarditis usually precedes pericarditis. The murmurs present must be called accidental if the area of cardiac dulness is normal, the second pulmonary sound not accentuated, and if the murmur be heard only at the pulmonary cartilage, or at this point and at the apex, and, at any rate, more distinctly at the pulmonary cartilage (Leube<sup>2</sup>). The elimination of *old endocarditis* or *chronic valvular disease*—a matter of importance—may be accomplished by attention to the character of the murmur in acute endocarditis, as well as to its limited area of diffusion, and by the absence of the signs of hypertrophy and of accentuation of the second pulmonary sound.

A *relative insufficiency* distinguishes itself by a pure systolic murmur, loud and not invariably uniform, by a weak cardiac impulse, a slight accentuation of the second pulmonary sound, and a comparatively small and often irregular pulse. It is met with in excessive dilatation of the left ventricle, in anemia, “and particularly in certain changes of the valvular muscles, due to myocarditis” (Leube).

**Prognosis.**—The immediate dangers are few, and depend largely upon the primary disease. In many instances, however, acute endocarditis initiates permanent lesions of the valves.

**Treatment.**—**Prophylaxis.**—The prevention of acute endocarditis in rheumatism has been dealt with in discussing the latter disease. No known direct measures can prevent the development of this condition in the course of the specific fevers, though absolute rest in bed and protection of the body against “cold” may diminish somewhat the tendency to it.

**The Attack.**—The sick-room should be free from draughts, though well ventilated, and flannel is to be applied to the chest. The diet may be liberal, but should be composed chiefly of milk and other light nutritious substances. Stimulants are required in most instances, and

<sup>1</sup> *Deutsch. Archiv f. klin. Med.*, Nov. 5, 1896.

<sup>2</sup> *Loc. cit.*



in abundance should the heart be failing. Digitalis is to be employed cautiously if at all. When the myocardium is involved, its use is not without danger; under these circumstances the drug increases the sufferings of the patient by throwing the inflamed and weakened cardiac muscle into firm contractions. The salts of ammonium, particularly the carbonate, should be given continuously with a view to obviating intracardial coagulation of blood; and should the latter accident occur despite all efforts to prevent it, the carbonate, together with strychnin and alcoholic stimulants, should be freely administered. I am convinced that in endocarditis due to acute articular rheumatism it is wise to continue the exhibition of the salicylates, though in moderate doses, provided that the heart is guarded by the use of stimulants. During convalescence from an acute endocarditis the patient should be kept at rest, so as to minimize the strain upon the affected valves; even after he has apparently recovered, and particularly should the murmur still be present, perfect quiet is to be enjoined for a period of several weeks.

#### ULCERATIVE ENDOCARDITIS.

(*Malignant or Infectious Endocarditis.*)

Malignant endocarditis is variously characterized, though usually either by perforative ulceration, by suppuration of the valves, or by both, giving rise to the physical signs of acute endocarditis. These develop amid the symptoms of a severe type of some primary infectious or septic disease. There is at hand sufficient clinical evidence to warrant the assumption that ulcerative endocarditis also occurs, though very rarely, as a primary affection.

**Pathology.**—(a) *Valvular Endocarditis.*—In its early development the valves are the seat of vegetations (such as are met with in simple acute endocarditis) which later undergo necrosis. The latter process manifests a tendency to spread, destroying more or less of the endocardium. In the interior of the vegetations the process of suppuration not infrequently takes place, and the abscesses thus formed rupture and produce various lesions according to their size and situation. The vegetations take on a grayish- or yellowish-green appearance. Histologically, they are composed of granulation tissue, veiled by granular and fibrillated fibrin that contains numerous micro-organisms. At the base there is usually developed more or less reactionary inflammation. After rupture the blood-current may enter the abscess-cavity, and, if there be no complete perforation, the endocardium will be pouched out, and an aneurysmal dilatation of the valve will result. Ulcerative lesions are most frequently observed. They may be mere erosions of the endocardium, but, as a rule, are penetrating in character and often result in complete perforation. I have seen repeated instances in which the three classes of lesions above depicted were all present. Osler, in an analysis of 209 cases examined by him with a view to ascertaining approximately the relative frequency with which the different parts of the heart were affected, obtained this result: Aortic and mitral valves together, 41; aortic valves alone, 53; mitral valves alone, 77; tricuspid in 19, pulmonary valves in 15, and the heart-wall in 33 instances. In 9 instances the right heart alone was involved.<sup>1</sup>

<sup>1</sup> *Text-Book of Medicine*, p. 631.

(b) *Malignant mural endocarditis* gives the same set of changes as the valvular form; indeed, the latter may be combined with the former throughout. It is a comparatively rare condition, as is shown by the foregoing figures of Osler. The ulcerative process may invade the chordæ tendineæ and the valves, and may perforate the septum or even the ventricular wall itself. The vegetations are detached in small or large masses, and are conveyed by the circulating medium to various distant organs, especially to the spleen and kidneys, less frequently the intestines, meninges of the brain, and the skin. Their site is determined largely by their size, and they may be so large as to plug vessels of the caliber of the external iliac. When found in the lungs they may originate in endocarditis affecting the right heart. These emboli, containing, as they do, the agents of inflammation, form suppurative infarcts that may be either white or red in color. The detached vegetations are sometimes so laden with irritants as to cause rapid softening of the coats of the vessel at the point where they become arrested, with consequent aneurysmal dilatation directly opposite their seat. As to number, the infarcts vary greatly in different cases; thus there may be only one or two, as in a case in my own knowledge in which the spleen alone contained two small infarcts, or there may be more than a thousand minute abscesses widely scattered throughout the body.

**Etiology.**—It is to be kept in remembrance that the condition is, with few exceptions, most probably a secondary one. The disease, therefore, arises in consequence of secondary infection, and this explains why the lesions peculiar to simple endocarditis usually precede and accompany those of the ulcerative form. The specific irritant is probably the *streptococcus pyogenes* (Fränkel and Sängner); and if this be true, the diseases in which ulcerative endocarditis occurs as a complication merely furnish the opportunity for the invasion of the streptococcus. The bacillus diphtheriæ, however, as well as the bacillus coli, the bacillus anthracis, the pneumococcus, the gonococcus, and other organisms, have been found in some cases in the absence of the streptococcus. In purely septic diseases ulcerative endocarditis forms but a part of the serious general condition. Here the cardiac element serves to facilitate the generation and rapid diffusion of the poison; and, since the latter is prone to attack the valve-segments, the morbid lesions within the heart not rarely constitute the chief pathologic factor in septicopyemia. Instances, however, are met with in which the segments present slight changes. The malignant form occurs, in connection with acute articular rheumatism, in about 10 per cent. of the cases in which acute endocarditis appears. Among many other diseases that furnish occasional instances of this serious complication are diphtheria, scarlet fever, typhoid fever, erysipelas, small-pox, chorea, tuberculosis, and chronic Bright's. In some of these, simple acute endocarditis, it will be remembered, occurs with relative frequency. In lobar pneumonia the ulcerative type is common, occurring almost as frequently as the simple variety, and was found by Osler in 11 out of 23 cases. The septic processes that arise from the puerperal state or from gonorrheal infection may also be complicated with ulcerative endocarditis.

**Clinical History.**—That form of ulcerative endocarditis which is a more or less prominent factor in septic diseases has been considered in



connection with septico-pyemia. Malignant endocarditis being usually a secondary event, its clinical features must not be confounded with those of the primary affection in the course of which it occurs. It is, however, often impossible to clearly separate the former from the latter, and the original disease often appears to be but slightly modified.

*Local symptoms* are often entirely wanting, or, when present, consist merely in slight precordial pain and oppression, and are not sufficiently well pronounced to arrest attention. Subjective symptoms are, however, connected with other organs than the heart, and are due to the irritating effects of emboli that occupy the various organs of the body. Gastro-intestinal disturbance, as shown by the occurrence of vomiting and diarrhea, is common. Pain that is ascribable to local peritonitis over the spleen, and sometimes also over the liver, is observed. Hematuria and dimness of vision are also frequent concomitants, and are due to renal and retinal hemorrhages. The urine may be scanty and albuminous. The more *general features* that are the result of the local embolic processes and, in part, of the valvular lesions, are serious and for the most part typhoid in character. The onset is usually signalized by a severe rigor that may be repeated at intervals varying from one to several days, and the disease presents an irregularly remittent temperature-curve, often touching a high mark ( $105^{\circ}$  or  $106^{\circ}$  F.— $40.5^{\circ}$  or  $41.1^{\circ}$  C.). I saw a case recently in which the febrile movement pursued the continued type for seven weeks. The pulse is rapid and irregular, though frequently becoming slow within a brief period. The patient rapidly emaciates, and from the earliest development is profoundly prostrated, and nervous symptoms, as headache, mild delirium, followed by somnolence, and sometimes even coma, appear. Profuse sweating sets in and persists, and as a result the skin may be covered by sudamina. An ecchymotic eruption due to cutaneous emboli is also common, this being often found associated with a papular or a diffused roseolar rash.

**Physical Signs.**—These may be negative as regards the heart. In the majority of instances, however, a systolic murmur is present, which, when associated with other clinical indications that point to this affection, is valuable for diagnosis, and especially so if developed while the patient is under treatment for the primary attack. The second sound is sometimes accentuated even when no organic lesions have previously existed. The physical signs of pneumonia and pleuritis (particularly the latter) may not infrequently be noted. Cases occur in which gangrenous infarcts of the right lung give rise to signs of localized consolidation; the spleen becomes considerably swollen, as can be easily demonstrated by palpation, and is quite tender as a rule; and the liver is likewise moderately enlarged and slightly sensitive.

*Cerebral Variety.*—In a small though decided percentage of the cases all the clinical features of acute suppurative meningitis are presented, and sometimes to the almost total exclusion of symptoms pointing to the primary disease or to the more typical typhoid form of ulcerative endocarditis. For a description of the symptoms that characterize the cerebral form the reader is referred to the discussion of Purulent Meningitis.

*Recurrent Malignant Endocarditis.*—By this term is meant an acute ulcerative endocarditis coming on in the course of chronic valvular disease. As has been pointed out, simple acute recurrent endocarditis is



common, though difficult of recognition. The latter condition, as well as the lesions in chronic valvular disease, predisposes to secondary infection by the streptococcus and other organisms. The onset is usually abrupt and marked by a chill. The patient has fever, which may be quite high ( $104^{\circ}$  F.— $40^{\circ}$  C., or over), and may present either the irregularly intermittent type or the truly intermittent. The latter is often associated with recurring chills. In either of the above groups the course is apt to be acute. In some cases the character of the pre-existing murmur is changed, becoming louder and more decidedly blowing; in many other instances, however, there is no appreciable alteration in the murmur. The condition may arise suddenly, amid the signs of failing compensation, as in a fatal case reported by Dr. H. P. Loomis,<sup>1</sup> in which the patient was semi-conscious, cyanotic, and suffering from intense dyspnea and general dropsy. It was impossible to diagnosticate the cardiac lesions by the murmur present. Occasionally these severe intercurrent febrile attacks end in recovery, and such cases probably belong to the benign form, though closely simulating the malignant in their clinical character.

There is a third class of cases that run a subacute or even chronic course, with more moderate elevations of temperature, or, as rarely happens, none at all. Mullin of Hamilton has reported a case that lasted more than a year. Here the other clinical phenomena, especially those referable to the heart, are often scanty and indefinite.

**Diagnosis.**—It is of paramount importance to consider the previous history and all the circumstances under which individual cases occur. These points, together with the symptoms attending the onset and the first three or four days of illness, more particularly the severe rigor, early high temperature, and profound prostration, the sweatings, the various embolic phenomena, and the presence of cardiac symptoms, are often adequate for a positive diagnosis. With a clear history and the presence of the more characteristic general symptoms (in particular, the signs of embolism), a correct diagnosis is possible, even though cardiac murmurs be absent. Instances in which no data can be found to explain the occurrence of the disease are especially puzzling, and these will remain unrecognized if at the same time the lesions in the heart fail to be manifested by special symptoms. The existence of a chronic valvular affection would, in itself, under the latter circumstances afford strong probability of the presence of recurrent malignant endocarditis if the other significant clinical symptoms above mentioned were present.

**Differential Diagnosis.**—There is a group of cases in which either the history fails to furnish the essential causal factors on the one hand, or there is an absence of definite heart-symptoms on the other; this group cannot sometimes be separated from cases of *typhoid fever*. The subjoined table will, I feel, be found valuable as an aid in eliminating the latter disease from the typhoid form of malignant endocarditis:

## ULCERATIVE ENDOCARDITIS.

## TYPHOID FEVER.

Previous or associated disease, as acute rheumatism or pneumonia.  
Very rarely a primary affection. No prodromes observable.

Health good before the time of onset of the attack. History of epidemic.  
Always idiopathic, with a prodromal stage.

<sup>1</sup> *Transactions of the New York Pathological Society*, 1890.

## ULCERATIVE ENDOCARDITIS.

Ushered in suddenly by a severe rigor, which may recur.

The fever rises rapidly.

Profound prostration as early as third day.

The fever is markedly irregular from time of onset, as a rule.

Embolic symptoms (hemiplegia, etc.) may appear.

Cardiac symptoms, especially loud systolic murmur, often present.

The blood usually shows signs of septic leukocytosis.

## TYPHOID FEVER.

Invasion marked by slight recurring chilly sensations. (Severe chill very rarely.)

More gradually, in step-like fashion.

Profound prostration not earlier than seventh day.

Less so, especially in the first week.

Extremely rare.

Sometimes a soft systolic murmur.

The blood shows a decrease in the number of leukocytes.

The *cerebral* form can only be distinguished from purulent meningitis due to other causes if there be a history of definite causal factors or if there be present distinct cardiac symptoms.<sup>1</sup>

**Prognosis.**—Most cases that run an acute course terminate in death, and when supposed instances of malignant endocarditis recover they are usually to be regarded as being of benign character. Subacute or chronic varieties, however, such as are most frequently met with in connection with organic heart-disease, sometimes reach a favorable issue.

**Treatment.**—This is largely supportive. The feeding is to be pushed vigorously, and concentrated forms of liquid food should be given at regular, brief intervals. Arterial stimulants in liberal quantities are also demanded, and in addition quinin and antiseptics may be tried. For the embolic symptoms the salts of ammonium give slight promise of beneficial results, and I prefer the carbonate for this purpose. Unfortunately, no known method of treatment is of any positive avail.

## CHRONIC ENDOCARDITIS.

(*Chronic Interstitial Endocarditis.*)

Two clinical varieties are met with—one following the acute form, the other beginning as a chronic inflammation.

**Pathology.**—The lesions may be limited to the valvular endocardium (their most common seat), or the mural endocardium may also be involved. In not a few instances the lesions are confined to the edges or bases of the segments, and when seen in the early stages there may frequently be observed merely a slight thickening of the free border of the leaflets: in most cases small prominences appear near their free margins. The endocardium looks opaque and its normal elasticity is lost quite early. When the auriculo-ventricular valves are affected the primary seat of inflammation is the auricular face, but when the semilunar valves are diseased the morbid changes begin on the ventricular side and implicate the Aurantian body. Extension of the morbid process to other and all parts of the valvular curtain is common, and it is in cases of this sort that the greatest degree of shrinking and crumpling occurs. The most characteristic lesions consist of inflammation and exudation, which produce cohe-

<sup>1</sup> The septic form may simulate malaria in its general course. The points of dissimilarity may be found in the discussion of Septico-pyemia.

sion of the segments, roughen the surfaces, and lead to the deposit of fibrin upon them. The histologic alterations consist in a proliferation of the endothelial and a round-cell infiltration of the subendothelial connective tissue. Organization of these products of inflammation into connective tissue, with resulting induration and contraction, is the necessary subsequent pathologic event. In old cases calcification of the diseased structure is frequent. The shrinking shortens the curtains or curls their free edges, and produces insufficiency in either case, since on dropping into the plane of the valvular orifice they fail to close it perfectly. Valves thus deformed may also obstruct the blood-stream. As before mentioned, cohesion of the invaded segments takes place, particularly at their bases, and may extend upward for a considerable distance, leading to constriction or stenosis.

Involvement of the semilunar (aortic) segments in the ways previously described opposes an obstruction to the outflowing blood-current on the one hand, and, owing to the inability of the segments to effect perfect closure of the aortic orifice, allows on the other hand a diastolic reflux of blood into the left ventricle. The aortic ring to which the semilunar segments are normally attached becomes sclerosed, and finally the seat of atheromatous changes, either fatty or calcareous. Again, chronic inflammation of the intima of the aorta produces a similarly thickened condition of this layer in spots, followed by atheroma. These changes are most prone to take place in the course of the ascending arch of the aorta or just above the aortic segments. The fact of really vital importance in this connection is that from the aorta and subvalvular ring the diseased processes before described may extend to the coronary arteries. Hence sclerotic and atheromatous alterations are found frequently in association with organic valvular defects. The great clinical significance of the implication of these vessels will be emphasized hereafter.

Much less commonly similar lesions are noted at the orifice of the pulmonary artery. A similar involvement of the auriculo-ventricular valves also causes regurgitant and obstructive deformities at the mitral orifice, and in advanced cases the chordæ tendineæ, and even the papillary muscles, are almost invariably invaded by direct extension from the valves. As these structures undergo marked thickening with subsequent contraction, they become shortened and rigid, causing an actual narrowing of the cardiac orifice. In mitral stenosis during the early stages or in the mildest types a more or less complete ring of vegetations encircles the mitral orifice on its auricular aspect. The margins of the orifice also become hardened and roughened, these changes frequently extending to the valvular curtains and the chordæ tendineæ. Under such circumstances the thickened valve could not, during the ventricular diastole, be forced back against the ventricular wall, but would occupy a nearly central position. Owing to cohesion of the free edges of the valvular structures and to contraction of the chordæ tendineæ drawing the leaflets toward the apex of the heart, the transition from this condition to the formation of a hollow cone (*funnel mitral*) is accomplished by natural, easy stages. Extensive union of the segments along their free margins may reduce the aperture to a mere button-hole slip (*button-hole mitral*) as viewed from the auricular aspect. The last two forms of lesions are far less commonly met with at the aortic orifice, though they occur rarely in



moderate degree; on the other hand, curling of the valvular edges is far more commonly seen at the aortic than at the mitral orifice, if we except the cases that occur in children. The curtains of the thick, rigid valves may also permanently occupy the plane of the orifice, presenting a small ring-like opening (*annular mitral*).

Fatty degeneration leading to the formation of necrotic (atheromatous) ulcers is common; and calcareous deposits are frequently seen in old cases, either in localized areas or coextensive with the diseased tissue, converting the entire valve into a calcified mass, with loss of the valvular outlines.

In *chronic mural endocarditis* the lesions exhibited are grayish-white, slightly elevated patches that are usually found to invade the underlying muscular structure to a greater or a less extent. Under such conditions of the valves the deposit of fibrin would be greatly favored, and the presence of an ulcerative surface or of a fibrous deposit on the valves affords a ready and satisfactory explanation of the occurrence of embolism in these cases. Emboli may also become detached from cardiac thrombi or from thrombi formed in the peripheral veins. For anatomic reasons the favored seats of embolic processes are, as in acute endocarditis, the spleen, brain, and kidneys, and irritants that cause acute endocarditis find here a tissue-soil whose capacity for resistance to invasion is greatly lowered. Chronic mural endocarditis and chronic myocarditis are, as a rule, due to the extension of the inflammation from the valves, though the ventricular endocardium may be invaded independently of the valvular affection. In one instance of mitral stenosis I observed an enormous calcareous mass partly in the subvalvular tissue and partly in the wall of the ventricle, the segments remaining altogether intact. In advanced stages of most cases of chronic endocarditis myocardial degeneration occurs. It takes the form of fibroid change or fatty degeneration, or both. Aortic-valve involvement, especially when complicated with atheromatous change in the coronary arteries, is most prone to these forms of myocardial disease. Chronic endocarditis may be said to persist until death. The effect of the valvular deficiencies that have been described upon the several cardiac chambers and the muscular structure of the heart will be most advantageously studied when the individual lesions of the segments are considered.

**Etiology.**—There can be no doubt that most cases of organic heart-disease occurring in children and young adults are caused by primary acute rheumatic endocarditis; and, although the latter affection cannot in truth be said to invariably terminate in chronic endocarditis, it probably does in most instances. This result, in my opinion, is more frequent in children suffering from acute endocarditis than in adults. On the other hand, not a few cases of chronic endocarditis originate in a very mild grade of acute valvular inflammation, which may be, though itself mute, reinforced by a rheumatic diathesis. Indeed, acute endocarditis may be the sole expression of rheumatic disease. Not less than one half of all cases of organic valvular disease are caused by rheumatism, and more than one half of the total number occur between twenty and thirty years of age. Acute endocarditis complicating other acute infectious diseases than rheumatism (*e. g.* measles, chorea, pneumonia) may also be followed by the chronic variety; but it is quite questionable whether this occurs as frequently as in the case of acute endocarditis of rheumatic origin.

The second variety, in which slow interstitial changes occur from the beginning, is dependent upon—(a) biologic irritants (*e. g.* syphilis, malaria, and chronic rheumatism); (b) chemical irritants (uric acid, alcohol, lead); and (c) mechanical influences. Doubtless the influence of repeated straining efforts is the most potent cause of this class of cases. Heavy muscular labor increases constantly the tension in the arterial system, and this acts injuriously upon the valve-segments, setting up a gradual sclerotic change. In like manner, *arterial sclerosis* and *Bright's disease* may cause chronic interstitial endocarditis by increasing constantly the vascular tension, though the fact that these affections may in turn result from the action of some of the leading causes of organic heart-disease must also be recollected. *Trauma* has produced in valves previously healthy a sudden, incontestable proof of valvular paresis or laceration, that has persisted in a few well-attested cases. This accident is of course much more frequent where the valves have been already diseased, and particularly if they have been the seat of lacerative processes.

The predisposing causes of organic valvular disease may be discussed briefly. *Hereditary influence*, as pointed out by Virchow, is especially potent in persons in whom there is hypoplasia of the heart and aorta (*e. g.* in chlorosis). It may be said that any malformation of a valve is certain to throw an undue strain upon certain portions, and hence is likely to be followed by interstitial change. Osler, in 17 cases of bicuspid aortic valve, has reported the segments to be uniformly sclerosed. The cases of supposed hereditary transmission are doubtless, however, for the most part, due to the causes before mentioned, and particularly to rheumatism. *Age* exerts a predisposing influence, its effects, however, varying with the valve implicated. During fetal life this is on the right side of the heart in a vast majority of cases; during childhood, adolescence, and early adult life, when the infectious diseases and rheumatism are frequent, it is the mitral valve in most instances; and finally, during middle and especially during advanced life the aortic segments are especially involved. I have, however, found aortic disease to be more common in young adults than most writers are ready to admit, and that it is favored especially by an occupation involving muscular strain (*e. g.* blacksmiths, draymen, soldiers during campaigns). *Sex per se* has little if any effect, though, owing to the greater frequency of certain well-known causes of valvular disease (chorea and rheumatism) in girls and young women, females are more frequent sufferers than males.

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## AORTIC INCOMPETENCY.

(*Aortic Insufficiency; Aortic Regurgitation.*)

**Definition.**—The failure of the aortic valves to prevent a return flow of blood into the ventricle, owing, as a rule, to a diseased condition of the aortic leaflets (sclerosis) that is followed by crumpling and attended with contraction, shortening, or curling of the edges, and finally calcification.

**Pathology.**—The aortic orifice may be enlarged (relative insufficiency), and here the normal cusps fail to effect complete closure of the



orifice. The flaps of the diseased aortic valves sometimes adhere to the intima of the aorta, and laceration of the semilunar segments, which are the seat of diseased processes (particularly ulceration), is sometimes found *post mortem*, and may be the chief factor in determining the development of the condition. This accident may very rarely occur as a result of a severe straining effort in the case of valves previously healthy. Occasionally, also, the chief factor in the production of this valvular lesion is a congenital malformation of the segments whereby they are rendered very prone to chronic endocarditis in consequence of the undue strain to which they are subjected. The lesions that give rise to stenosis may coexist with simple aortic incompetency, and, though the latter condition frequently occurs alone, stenosis is quite as often combined with regurgitation.

**Mechanical Influence of the Lesion.**—The reflux current passes from the aorta backward through the imperfectly closed semilunar valve into the left ventricle during the diastole of the heart or while the left ventricle is being filled by the normal blood-flow from the auricle. It is clear that over-distention of the left ventricle must result at once from two simultaneous influx currents of blood, with a tendency to an increasing dilatation, especially since the lesion itself is steadily progressive. To expel the increased amount of blood from the left ventricle demands increased cardiac power, and the over-exertion causes dilatation, followed by a compensatory hypertrophy. Dilatation and hypertrophy of the left ventricle develop *pari passu* until the left ventricle reaches enormous dimensions, forming the *cor bovinum*, which weighs 1000 grams or more (30 to 50 ounces). Under these circumstances the arterial system is overfilled at each ventricular systole. In the very early stage the reflux of blood from the aorta into the ventricle tends to lessen the volume of the circulating medium in the arterial tree, but this depleting influence is successfully counterbalanced by the augmented column of blood thrown from the ventricle during cardiac systole. Hence the requirements for bodily nutrition are, for a longer or shorter time, satisfied. The abnormally large amount of blood that is thrown into the arteries with undue force subjects them to increased tension, and as a result arterio-sclerosis, leading sometimes to atheroma, is commonly developed, and presents its ulterior dangers (aneurysm, apoplexy). The coronary arteries are similarly involved, their caliber being reduced, and particularly at the point of origin. Soon or late the blood-supply to the heart-muscle may become inadequate, and nutritional disturbances now manifest themselves in fatty and fibroid degeneration of the cardiac muscles; these pathologic changes are attended with secondary dilatation, which soon predominates over the hypertrophy. The imperfect blood-supply to the ventricular tissue may be accounted for, in great measure, by the narrowed lumen of the coronary vessels, and also in part by the inelasticity of the walls of the latter and by the inefficiency of the aortic recoil. Furthermore, it is to be recollected that, in obedience to the laws of nature, overuse of any single group of muscles, while productive of marked hypertrophy in the first instance, is followed eventually by atrophy and loss of power. In consequence of the increased tension to which they are constantly subjected the mitral leaflets may become the seat of sclerotic endocarditis, and this may lead to the development of mitral insufficiency (usually of



mild grade); or there may be a displacement of the mitral segments in the direction of the auricle, thus creating incompetency at this orifice. There is to be observed in many instances a marked degree of fatty degeneration of the papillary muscles, which also exhibit more or less flattening. Again, *secondary dilatation* may produce relative insufficiency at the mitral orifice. When incompetency has been established here, impeded pulmonary and general venous circulation, together with the secondary lesions in the left auricle, pulmonary vessels, and right ventricle that are characteristic of that valvular lesion, are the necessary result. The blood-current through the mitral ring may be retarded, owing to the simultaneous influx into the left ventricle from the aorta, thus causing pulmonary congestion without organic change in the segments.

**Special Etiology.**—(1) *Acute Endocarditis*.—Incomplete resolution of the acute form of endocarditis leads to progressive chronic valvular disease. In the young it is caused with comparative frequency by rheumatic endocarditis. Thus, aortic regurgitation may arise, though rarely, in the course of acute endocarditis, as, for example, when the latter is attended with destructive ulceration. Such instances usually terminate in speedy death.

(2) *Chronic Infectious Irritants*.—I have found syphilis to be a factor (though rarely the sole cause) in a considerable percentage of cases. Aortic regurgitation is a frequent complaint in sailors and soldiers, among whom it is worthy of notice that syphilis is particularly common.

(3) *Chemical Irritants*.—(a) *Uric Acid*.—In chronic and irregular forms of gout the irritating qualities of uric acid give rise to interstitial endocarditis and arterial sclerosis. It is quite probable that chronic rheumatism has a similar influence, though brought about in a somewhat different manner. (b) By favoring the accumulation of uric acid in the blood, *lead-poisoning* may be indirectly responsible for the disease. (c) *Alcohol* by its irritant action may excite chronic valvulitis.

(4) *Augmented Aortic Tension*.—The excessive functional activity of the heart occasioned by the immoderate use of cardiac stimulants (alcohol) tends to raise the blood-pressure above the normal point, and thus sclerotic endocarditis may be developed very slowly. The effect of *occupation* in causing this disease, by increasing the vascular tension, is more notable than in the case of alcohol, though both of these factors are found not infrequently to be present in the same case. It is undeniably true that strong-bodied men in the middle period of life and those engaged in heavy manual labor are the most frequent sufferers from organic disease of the heart, and that such occupations as demand the repeated putting forth of strong efforts are powerful factors in causing aortic incompetency.

(5) From personal observation I feel convinced that chronic endocarditis (affecting the aortic valves) may be secondary to *aortic endarteritis* as the result of direct extension. It must be borne in mind, however, that arterio-sclerosis is also often secondary to chronic valvulitis.

(6) Relative insufficiency is caused, in rare instances, by pronounced dilatation of the ascending portion of the arch near to the valve, or by an aneurysm just beyond the aortic orifice.

Among the more effective predisposing factors are *age* and *sex*. The disease occurs much more often in males than in females, chiefly on

account of the fact that a greater percentage of the former than of the latter are engaged in occupations that are causally related to the disease. As to age, a preponderating proportion of the cases arise during advanced middle life, and a comparatively smaller number at a more advanced period than in young adult life.

**Symptoms.**—So long as the hypertrophy of the left ventricle successfully overcomes the otherwise injurious consequences of the valvular defect the harmonious balance of forces is maintained, and there is an almost entire absence of symptoms. I have observed, moreover, that compensation does not fail so early in young subjects as in those more advanced in years, this being probably due to the fact that the disease is frequently associated with, or secondary to, atheromatous changes at the latter period of life. With the development of marked hypertrophy severe muscular exertion and strong mental excitement will, by exciting over-action of the powerful heart, bring on a train of symptoms, as throbbing headache, vertigo, and tinnitus aurium. The clinical manifestations of arterial anemia, particularly of the brain, and also those of general arterio-sclerosis, frequently coexist. The patient's countenance exhibits pallor, and he complains of headache, flashes of light before the eyes, and dizziness. Dilatation of the peripheral vessels often leads to hot flushes and drenching sweats. Cases exhibiting the latter symptoms have been mistaken for phthisis. Dizziness is often distressing, and is most marked upon rising from the recumbent to the erect posture. Shortness of breath may come on early, but this rarely happens except upon inordinate exertion or great mental excitement—conditions that cause strong cardiac action and prohibit the discharge of blood from the left auricle into the left ventricle, thus causing *pulmonary congestion*. Oppression in the precordial region and cardiac palpitation are commonly present, as is a dull aching pain; the most constant seat of the latter is the precordia, but it radiates not infrequently to the shoulders, and thence down the arms, particularly the left. Genuine *angina pectoris* may be a concomitant. I have also seen a couple of instances of aortic regurgitation in which severe pain was located in the left shoulder-joint, the condition simulating very closely rheumatism, though the latter affection could be readily excluded both as a causal factor and a complication.

Following immediately upon failure of compensation the cardio-pulmonary circulation is retarded, and there is increased *dyspnea*, the latter symptom being greatly intensified by undue exertion. There may be cough, and not rarely hemoptysis, though less frequently than in simple mitral disease. Later on, *general venous congestion* of a moderate grade follows pulmonary congestion, and the dyspnea now becomes severe. It is nocturnal, and often compels the patient to assume a semi-erect posture in bed. In the later stages the symptoms are due to mitral incompetency, followed by failure of compensation. *Edema* of the feet appears, and rarely goes on to general anasarca. In aortic incompetency a higher grade of symptomatic anemia is reached than in any other cardiac lesion—a recent blood-count showing 2,800,000 red corpuscles to the c.mm. Hence slight edema of the feet may be due solely or in part to anemia. The intercurrent of acute endocarditis, as evidenced by prostration and



irregular fever, is observed, and not infrequently as a terminal condition. The symptoms of cerebral, splenic, and renal *embolism* may arise. Probably sudden death ensues, as the result of involvement of the coronary arteries, with greater frequency in this than in all other forms of valvular disease combined; and yet this accident is by no means of frequent occurrence. Instances of aortic incompetency, in which *nervous phenomena*, as peevishness, irritability, or melancholia, manifest themselves, are too common to be looked upon as mere coincidences. Many patients are doubtless led to commit suicide because of their cardiac lesion when other and erroneous explanations are given to account for their acts.

**Physical Signs.**—*Inspection* brings to light an enlarged area of the apex-beat; this is displaced downward, being visible in the sixth and seventh interspaces and to the left, and most marked between the mammary and anterior axillary lines. The entire precordial zone may be distended, particularly in young subjects, and the systolic pulsation is usually more or less heaving in character. The carotids throb forcibly, as do the temporals, brachials, and radials, though less violently. These abnormal pulsations are due chiefly to hypertrophy of the left ventricle, though frequent factors of lesser influence are associated—an arterio-sclerosis and a regurgitant blood-stream from the aorta into the left ventricle. The impulse becomes diffused and wavy with the progressive enfeeblement of the left ventricle, and venous pulsation due to tricuspid insufficiency may be associated with arterio-pulsation later in the affection. Epigastric throbbing may also be noticed, and on gently rubbing a spot upon the forehead an alternate paling and blushing appear (*Quincke's capillary pulse*); this may also be noted in the finger-nails. It is not peculiar to aortic insufficiency, however, and may be observed in cases of decided neurasthenia and in anemia. Very rarely the pulse-wave is propagated from a capillary to the veins of the hand and back of the foot, giving rise to a visible venous pulsation. L. Webster Fox informs me also that the retinal vessels are seen to pulsate quite commonly in this disease.

On *palpation* a forcible heaving impulse is usually felt. When, however, dilatation predominates over hypertrophy, the impulse is weak and undulating. A diastolic thrill just to the left of the mid-sternum may be detected in many instances, and a presystolic thrill is also discoverable very rarely. The pulse is characteristic; it is quick, jerking, and full, but, upon striking the finger, recedes abruptly, and is known as the *Corrigan* or *water-hammer pulse*. This sudden collapse of the pulse is most decided when the arm is held in a vertical position. Its distinctive characters are not always appreciable after compensation is lost. A glance at the sphygmographic tracing will show a sudden rise and fall, with absence or delay of the secondary wave (*vide* Fig. 50).

**Percussion.**—Cardiac dulness is coextensive with the impulse, extending downward to the eighth rib, and to the left as far as, or even beyond, the anterior axillary line. Later, enlargement of the left auricle may cause dulness upward and to the left of the sternum. Enlargement of the right ventricle causes an increase of dulness to the right. When the dilatation exceeds the hypertrophy the area of dul-



ness will be much extended transversely and slightly upward, the apex now being more rounded.<sup>1</sup>

On *auscultation* a diastolic murmur becomes audible below and to the left of the aortic cartilage over the mid-sternum, and down along its left edge; this is produced below the aortic valve and in the left ventricle. From the xiphoid it is transmitted to the left as far as the spinal column. It may be heard in the vessels of the neck and, very

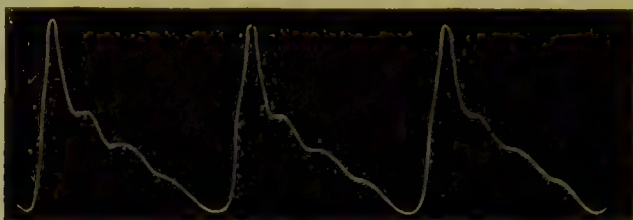


FIG. 49.—Normal pulse-tracing.

rarely, in the radials; occasionally its seat of greatest intensity is at the aortic cartilage, and, rarely, at the apex, as in a case under my own care at present. The rhythm of the murmur can be most readily de-

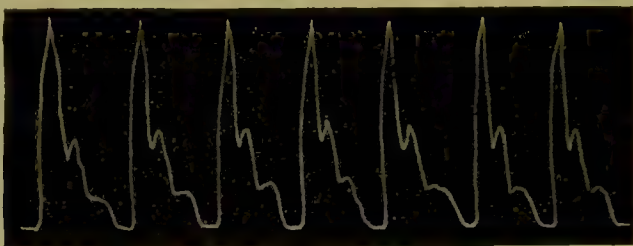


FIG. 50.—Pulse-tracing in a case of aortic regurgitation (William Hoffman).

terminated by ausculting over the base, for while the pulmonic second sound is usually audible at the apex (the murmur appearing to follow it), it is not so when, as sometimes happens, the murmur is quite loud. The first sound is often dull, indefinite, and widely diffused, owing to left ventricular hypertrophy. In quality this murmur is usually soft in character, blowing (long-drawn), and frequently musical; sometimes, however, it is somewhat rough and loud. In most instances a systolic murmur, brief and harsh in character and transmitted into the vessels of the neck, is also discovered over the aortic region (double aortic). The presence of the murmur with the first sound is not diagnostic of actual aortic stenosis. It is more often due to a mere roughening of the semilunar segments or of the intima of the aorta. In advanced cases a soft systolic murmur is commonly heard at the apex; it is readily distinguished from the diastolic murmur by its rhythm, and is occasioned in most instances by a relative mitral incompetency. Still another murmur, of rare occurrence, is rolling in character and generally presystolic in time, and may be heard at the apex over a limited surface-area. This may be accounted for by the presence of excessive dilatation of the left

<sup>1</sup> A dilated aorta with thickened walls—a condition sometimes associated with aortic regurgitation—may also give rise to abnormal dulness under the manubrium and to the left of the sternum.

ventricle, in consequence of which the mitral leaflets must remain free in the blood-stream during the diastole, and here they set up vortiginous movements that cause the presystolic (Flint) murmur. Duroziez discovered a double murmur in the arteries (femoral), which is quite frequently present, and, in view of its duplex character, possesses considerable diagnostic import. Traube has described another arterial phenomenon of interest—a systolic sound in the leg, somewhat resembling a heart-sound, but exceedingly short and sharp. It is probably due to sudden systolic distention of vessels that were previously empty.

The **diagnosis** demands the presence of a diastolic murmur, the signs of left ventricular hypertrophy, the peculiar arterial pulsations, and the characteristic water-hammer or Corrigan pulse.

The differential diagnosis will be considered in connection with the description of those complaints with which aortic incompetency is apt to be confounded. (See Aneurysm of the Arch, Hypertrophy, Dilatation of the Heart, etc.)

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## AORTIC STENOSIS.

**Definition.**—A narrowing or stricture of the aortic orifice, due to thickening or adhesion of the valve-segments, and causing an obstruction to the flow of blood into the aorta.

Simple aortic stenosis may be met with, though it is a great rarity. Its development is soon followed by more or less valvular incompetency, and hence these affections often coexist. It may be secondary to aortic insufficiency, but only rarely, the conditions in the latter disease being unfavorable to the development of the former.

**Special Etiology.**—Rarely rheumatic endocarditis, and still less frequently other forms of acute endocarditis, cause union of the semilunar segments, with resulting stenosis. The most common immediate causal factor is a slow sclerosis of the aortic valve, followed by calcareous deposits. The more or less immobile, rigid valves obviously narrow the aortic orifice and oppose a barrier to the outflowing blood-current from the left ventricle into the aorta. The aortic ring may be the seat of changes similar to those just described, resulting in a moderate grade of stenosis, though the leaflets themselves remain intact. The lesions are most frequently to be regarded as a part of the general process of arterial sclerosis, which is most marked in the region of the thoracic aorta; and sometimes, as Peter contends, they are distinctly secondary to sclerotic changes at the root of the aorta. The coronary arteries may be the seat of changes similar to those noted in aortic regurgitation. The condition is also rarely congenital. *Males* who have reached advanced years are especially prone to aortic stenosis, for the reason that atheromatous processes belong peculiarly to that sex and period of life.

**Mechanical Influence of the Lesion.**—To propel the normal volume of blood through the constricted aortic orifice requires increased strength on the part of the left ventricle, and, as a consequence, the latter hypertrophies. This hypertrophy develops very slowly, and keeps pace with the

progress of the valvular lesions. The undue ventricular tension sometimes induces more or less sclerotic change in the mitral valves. Hypertrophy of the left ventricle eventually gives way to extreme dilatation, and also to relative mitral incompetency with its unfavorable influence, first upon the pulmonary and, secondly, upon the general venous circulation.

**Symptoms.**—The symptoms date from the commencement of failure of compensation, often many years after the onset of the disease. Their first appearance will be found to follow some unusual muscular effort or the operation of some depressing influence, as the too free use of tobacco or alcohol. They are due to disturbances of circulation arising from a gradual secondary dilatation of the left ventricle, which is now unable to propel the normal quantity of blood into the arterial tree. Hence *anemia*, especially of the brain and peripheral parts of the body, becomes pronounced, and is evidenced by such symptoms as syncope, dizziness, headache, and pallor. Since aortic incompetency usually manifests itself secondarily, the clinical features of both affections are sooner or later variously commingled. In cases in which mitral lesions develop they are overcome by compensatory enlargement of the right ventricle: the latter chamber may then become dilated secondarily, in which event tricuspid regurgitation and the symptoms of general venous engorgement appear. As in the case of aortic regurgitation, so in an aortic constriction, *slight edema* of the feet is common as a terminal symptom; marked dropsy, however, is uncommon. From the fibrous deposits on the segments, as well as from any small clots behind the valves, emboli are apt to become dislodged by the forcible blood-stream and be conveyed to the brain (cerebral embolism), to the spleen (splenic embolism), to the kidneys (renal embolism), or to other organs.

**Physical Signs.**—*Inspection.*—The apex-beat is gradually displaced downward and to the left, owing to left ventricular hypertrophy. It is, as a rule, slow, forceful, and heaving, but less frequently it may be lacking in strength. It may be enfeebled, diminished in area, or even absent, owing to associated emphysema.

*Palpation* discloses the forcible and heaving impulse-beat, unless emphysema be present, when the heart and its movement may be concealed and the apex-beat become impalpable. A marked systolic thrill,

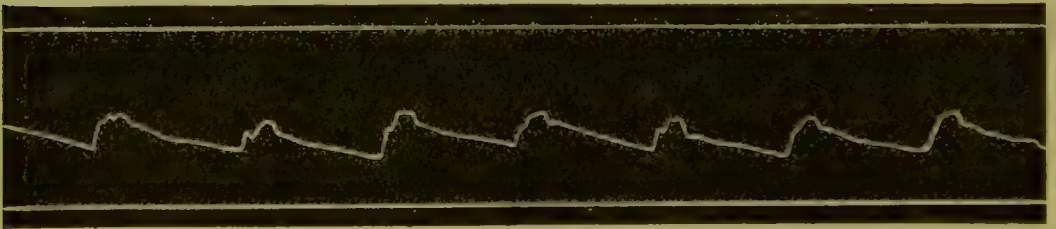


FIG. 51.—Sphygmogram of aortic stenosis, from a man aged sixty years.

with the seat of greatest intensity in the aortic region, is quite generally present. I have frequently felt this thrill in the apex region, though not so intensely as at the base. The pulse, in this disease, is small, regular, not compressible, and of normal or slightly lessened frequency. The sphygmographic tracing shows slowness of the ascending curve and a gradual formation of the descending line (*vide* Fig. 51).



*Percussion.*—Though there is developed in all cases hypertrophy of the left ventricle, the area of cardiac dulness is almost entirely dependent upon the degree of emphysema, if any be present. In the absence of this condition the dulness is increased to the left and downward, and especially so when insufficiency of the valve supervenes.

*Auscultation.*—A systolic murmur, harsh in quality, most audible at the aortic cartilage (the second right), and transmitted into the carotids, is present in typical aortic stenosis. When non-compensation is advanced the murmur is neither so rough nor so loud, and quite late it may be missing altogether. As aortic incompetency is commonly associated, a regurgitant or diastolic murmur is also heard, forming a double or seesaw murmur, the stenotic bruit more or less completely masking the regurgitant. A soft, blowing apical murmur (with the systole) is not infrequent in the advanced stage or after relative insufficiency of the mitral valves has appeared. The second sound is faint or inaudible on account of the diminished blood-tension in the aorta and the character of the valvular lesion.

The **diagnosis** demands the concurrence of the following signs: a systolic thrill, most marked at the base; a tense, small, somewhat slow pulse; indications of left ventricular hypertrophy (unless emphysema be present); a rough, loud, systolic murmur at the aortic cartilage and propagated into the vessels of the neck.

**Differential Diagnosis.**—A calcareous plate lying on the intima of the aorta and a markedly roughened condition of the aortic segments are conditions frequently mistaken for aortic stenosis, since they give rise to a murmur possessing many of the characteristics of the one above described. These murmurs, however, are seldom musical, while the murmur of aortic stenosis is often distinctly so; moreover, the second sound is decidedly accentuated, while in aortic stenosis it is faint or absent. In *chronic Bright's disease* with arterial sclerosis and left ventricular hypertrophy a murmur of maximum intensity may be developed at the base; but here the urinary symptoms, together with intensification of the second sound, are sufficient to establish a positive discrimination. In aortic regurgitation a systolic murmur frequently co-exists, but it cannot be reckoned as indicating actual stenosis unless it has a musical quality and unless a systolic thrill can be felt on palpation. In *chlorosis* and other forms of anemia basic murmurs are constant concomitants; the anemic murmurs are soft and distant, and not harsh; the intense thrill and hypertrophy are absent also. The venous hum may also be heard in the veins of the neck.

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## MITRAL INCOMPETENCY.

(*Mitral Regurgitation; Mitral Insufficiency.*)

**Definition.**—Imperfect closure of the mitral valve due to rupture or contraction of the mitral leaflets. It is also caused by dilatation of the left ventricle and by a diseased condition of the chordæ tendineæ.

**Pathology.**—This is the most frequent form of organic disease of

the heart. Thomas G. Ashton,<sup>1</sup> from clinical observation of 1012 cases of heart-affection, comprising all the different varieties, found that 54.4 per cent. were instances of mitral regurgitation. The predominating lesions are of three kinds: (*a*) Acute or chronic endocarditis, leading to contraction and deformity, particularly curling, of the margins of the valve; (*b*) contraction (shortening) of the chordæ tendineæ; and (*c*) relative insufficiency from excessive dilatation of the left ventricle (the segments being healthy). Adhesion of a segment with the walls of the ventricle occurs rarely, but may result in incompetency.

**Mechanical Influence of the Lesion.**—The mitral leaflets normally close, and prevent the reflux of the blood from the left ventricle into the left auricle with each cardiac systole. Hence incomplete closure of the mitral segments allows a portion of the blood to return into the left auricle during the systole. This regurgitant wave meets and offers an obstacle to the normal blood-current coming simultaneously from the pulmonary veins into the left auricle. It is clear that vortiginous movements must result under these circumstances and give rise to a murmur. The double blood-current, entering the left auricle during the systole of the left ventricle, causes over-filling (hence dilatation) of the left auricle, and thus induces compensatory hypertrophy of the left auricle, since its labor has been increased. During the next diastole the abnormally large contents of the auricle are passed under increased pressure into the left ventricle, producing over-distention (dilatation) of that chamber. This increased volume of blood in the ventricle is not all expelled into the aorta, but a portion of it returns into the left auricle. Thus the left ventricle, in consequence of its increased labor, becomes hypertrophied as well as dilated. Under these circumstances the volume of blood that is poured into the aorta remains about normal, and hence the arterial tension for a longer or shorter period is also normal. Soon the cardio-pulmonary circulation becomes impeded. The blood that returns into the left auricle must, by reason of pressure, offer increased obstruction to the overflow of blood from the pulmonary veins, and the pressure in the latter must, in turn, be similarly increased. The current of the blood through the pulmonary capillaries and branches of the pulmonary artery is thus retarded, owing to extension of the process of over-filling in a backward direction. The walls of the lung-vessels are the seat of a sclerotic process, and present an abnormal obstacle to the passage of the systolic wave from the right ventricle to the distal end of the cardio-pulmonary arc; in consequence of this the right ventricle becomes dilated and hypertrophied. The abnormally increased tension in the pulmonary vessels is shown by the accentuated pulmonic second sound. Thus the right heart compensates the lesion in the left, though to supply an adequate amount of blood to the peripheral arteries the left ventricle must maintain its proper degree of hypertrophy. As soon as this harmonious balance is disturbed, either as the result of increase in the degree of incompetency or of failure of muscular power, the progress of the blood from the right auricle to the right ventricle is hindered. Increased pressure in the right auricle produces dilatation of its chamber, with subsequent general venous congestion as a natural backward effect (*vide* Tricuspid Regurgitation). It is now seen that

<sup>1</sup> *Medical News*, June 30, 1894.



when the right heart fails a lessened amount of blood reaches the left ventricle, and hence an abnormally small amount finds its way into the aorta; this fact explains the presence of the low arterial tension late in the disease. Hypertrophy of the left ventricle in this disease has also been attributed in part to the augmented tension in the general capillary vessels that is occasioned by the venous stasis.

**Special Etiology.**—(a) *Rheumatic endocarditis* is the most frequent cause, though mitral regurgitation also results less frequently from acute endocarditis due to other causes. (b) It may be a part of a *general arterio-sclerotic process*, this group of cases being caused, not rarely, by syphilis and alcohol. (c) *A diseased condition of the columnæ carneæ or chordæ tendineæ*, if it weakens their structures so as to allow the free edges of the segments to pass beyond the plane of the orifice, produces mitral insufficiency. (d) It may arise in the course of *aortic valvular disease* (a secondary mitral affection), and is then excited mainly by undue tension of the blood in the left ventricle. Here the lesion is of a mild grade, as a rule. (e) It is frequently occasioned by *enlargement of the left auriculo-ventricular ring*, resulting from excessive dilatation of the left ventricle, as in aortic incompetency, aortic stenosis, long-continued fevers, and the graver anemias (relative incompetency). (f) *Ulcerative endocarditis*, either by perforating or producing rupture of the valve-curtains or by destroying the chordæ tendineæ, may bring about mitral incompetency. Among *predisposing factors* age and sex are worthy of special mention, the incompetency occurring with greatest relative frequency in young adults (from twenty to thirty years of age, according to Ashton's figures), and somewhat more commonly in males than females.

**Symptoms.**—*During Compensation.*—In healthy persons the compensatory forces keep pace with the valvular lesions for an indefinite and usually lengthy period, during which time there may be an entire absence of symptoms. When present they are dependent upon disturbances of the cardio-pulmonary circulation that are occasioned by trivial causes, such as excitement, going up stairs, or other forms of active physical exertion. Under these circumstances the force of the regurgitant current is increased (by the hypertrophied left ventricle), thus producing more or less pulmonary congestion that may proceed to edema of the lungs or hemoptysis. The condition is usually a temporary one, and is attended by dyspnea, palpitation of the heart, a short, hacking cough, and expectoration of a frothy serum that may be blood-stained. The relation existing between the severity of the dyspnea and the degree of active physical exertion is positive and vital. Shortness of breath may be the sole feature during a long period. The rational symptoms rarely warrant a suspicion of the existence of mitral disease until compensation has failed, but the patient's pulse often indicates heart-disease. The face is pale and the features peaked, the eyes, lips, and ears are dusky, and the minute vessels of the cheeks are prominent. Clubbing of the finger-nails is observed most frequently in the young.

*After Failure of Compensation.*—Failure of compensation implies failure of the right ventricle to force the normal quantity of blood through the left heart, with accompanying congestion of the lungs caused by engorgement of the systemic veins. The latter process begins at the right heart and proceeds toward the periphery, involving



the viscera, mucous membranes, and extremities until it is universal. The pulmonic symptoms above detailed are now more marked, particularly the dyspnea (which may be constant), cough (with expectoration of alveolar epithelium containing brown pigment-granules), and cardiac palpitation with arrhythmia. Pain is rare unless stenosis coexists. General venous engorgement manifests itself by an enlargement of the liver and of the spleen, in the features of gastro-intestinal catarrh, in hemorrhoids, in marked cyanosis of the surface, and in the passage of a scanty albuminous urine containing tube-casts and blood-corpuscles. Dropsy follows, beginning in the feet and progressing upward, until finally the trunk and the serous sacs are involved. By stimulation the heart may be reinforced, and all of the unfavorable symptoms disappear in consequence, but this is not for long, as a rule. I have at present under observation a case in which not less than half a dozen instances of broken compensation have occurred at intervals of six to eight months, all of which have been successfully overcome.<sup>1</sup> All instances prove fatal, and there comes a time when compensation cannot be restored and the end is reached by an uninterrupted downward course.

**Physical Signs.**—*Inspection.*—The precordia is prominent, particularly in children, and the area of the apex-beat is enlarged, later becoming diffuse and wavy. It is carried to the left and downward, corresponding with the degree of hypertrophy of the left ventricle. A pulsating epigastrium is in frequent association, particularly after dilatation of the right ventricle appears. With the failure of the right heart also come wavy pulsations in the cervical veins, and occasionally a mild grade of jaundice.

*Palpation* sometimes discovers a thrill at the seat of the apex-beat, that is synchronous with the first sound. The impulse during the stage of full compensation is forceful and heaving, but with the beginning of failure of compensation it grows feeble and irregular, and late in the affection is excessively weak and arrhythmic. The pulse bears a definite relation to the apical impulse. Thus it is regular and full during the compensatory period (though at times the tension is slightly lowered), but becomes small, easily compressible, and exceedingly irregular during the period of broken compensation. The latter pulse-characteristics become especially pronounced near the close.

*Percussion.*—The dull area is increased to the left, extending frequently to the anterior axillary line; and also to the right, frequently from  $\frac{1}{2}$  to 1 inch (1.2–2.5 cm.) beyond the right sternal margin. Dilatation of both ventricles exerts a widening influence; hence cardiac dulness is increased more laterally than vertically.

*Auscultation* reveals a systolic murmur, which exhibits its greatest intensity at the apex (see Fig. 52). Unquestionably, this murmur is also conducted, though rarely, to the tricuspid and pulmonary valves. Balthazar Foster first called attention to the fact that the murmur of mitral regurgitation may be loudest at the base of the heart and at times audible only in that situation—an occurrence that has since been confirmed by a number of authorities. It is sometimes audible in the recumbent posture and inaudible in the erect. From the apex

<sup>1</sup> Neglect of hygienic precautions, and intercurrent complaints of various sorts, often determine the occurrence of failure of compensation.

it is transmitted to the left as far as the angle of the scapula, with progressively diminishing clearness. It has a blowing quality, and frequently ends in a musical tone. Over the third left costal cartilage, and frequently at the apex, there is heard the accentuated pulmonic second sound, due to the increased tension in the pulmonary vessels that is engendered by the hypertrophy of the right ventricle. Combined murmurs may be heard, and not infrequently a rough, rolling, or rumbling presystolic murmur is detected. A frequent late occurrence is the secondary dilatation of the right ventricle, causing relative tricuspid insufficiency with its characteristic soft, low-pitched, systolic murmur, heard best at the ensiform cartilage. A spurious diastolic murmur may

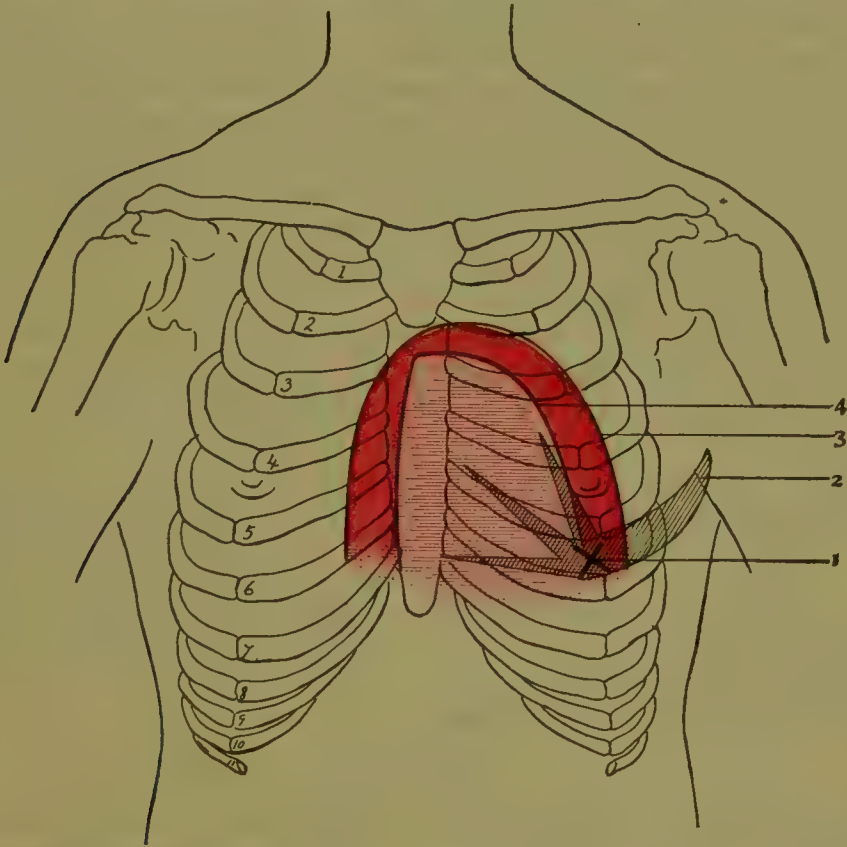


FIG. 52.—1, Seat of greatest intensity; 2, direction of chief transmission; 3, boundary line of relative dulness; 4, boundary-line of absolute dulness (modified from Sahli).

be noted, though rarely, when the sounds are timed with the pulse. This is due to the fact that occasional systoles are too weak to cause a radial pulse.

**Diagnosis.**—In the presence of the following group of features the diagnosis is set at rest: A marked broadening of the area of cardiac dulness; a systolic, *apical* murmur that is conveyed to the left axilla and may be heard even at the back; and a decided accentuation of the pulmonary sound. Obviously, the latter sound becomes feeble after dilatation of the right ventricle has occurred. A systolic thrill is of the highest diagnostic importance, but is unfortunately absent in perhaps a majority of the cases. Free regurgitation through the mitral orifice may be safely inferred when the following signs are concurrent:

(a) An absence of the sound of mitral-valve tension, a murmur replacing the first sound; (b) accentuation of the pulmonic second sound; (c) an enlarged area of the left cavity; (d) an enlarged area of the right cavity (Sansom).

**Differential Diagnosis.**—There are two organic lesions of the heart that are sometimes mistaken for mitral incompetency, since both are accompanied by a systolic murmur—the one *aortic stenosis*, and the other *tricuspid regurgitation*. How to distinguish mitral from tricuspid incompetency is a question that will receive due attention when the latter disease is considered. *Aortic stenosis* generates a systolic murmur, but it is loudest over the base, and is transmitted through the great vessels of the neck; while the mitral systolic is most intense over the apex and is transmitted far to the left. In mitral incompetency the pulmonary second sound is accentuated; in aortic stenosis it is not. In mitral incompetency both ventricles are enlarged, as shown by percussion and other signs; in aortic stenosis the left is chiefly enlarged during almost the entire course. In mitral incompetency a thrill, most marked over the apex-beat, may be felt; in aortic stenosis a thrill, rough and having its chief seat at the base, is common. Other minor points of distinction are furnished by the peculiarities of the pulse, the age of the patient, and other etiologic factors.

*Functional and other harmless murmurs* are often confounded with mitral insufficiency. The considerations on which the greatest dependence is to be placed in the differentiation are to be found in the subjoined table:

#### MITRAL INCOMPETENCY.

#### FUNCTIONAL AND HARMLESS MURMURS.

##### *History.*

Previous history of rheumatism or other disease causally related.  
Frequently there is definite knowledge of rheumatism and organic heart-disease, in combination in the same individual.

History of causal factors of one or other form of anemia, or of Graves' disease.  
No such association.

##### *Physical Signs.*

*Inspection.*—Dusky lips, ears, etc.; later wavy pulsation in veins of neck.

*Palpation.*—Finger-tips placed over apex-beat forcibly lifted. Pulse-tension somewhat lowered and not prolonged. Impulse displaced.

*Percussion.*—Evidence of dilatation of both ventricles.

*Auscultation.*—A systolic apex-murmur (often musical), with characteristic area of transmission.

Pallor of skin and mucous surfaces common.

Finger not lifted by the impulse, which often cannot be felt. Pulse-tension prolonged and arterial pressure increased generally. Impulse not displaced.

Dilatation of right auricle, but only in about one-half of the cases, giving rise to dulness above or to the right of the right edge of sternum.

Soft systolic murmur at apex (may be, though rarely, transmitted to axilla), usually preceded by or associated with a basic systolic murmur and a venous hum in the veins of the neck.

To differentiate the murmur of relative mitral incompetency is difficult, though in many instances it can be accomplished with reasonable certainty. It rests upon two points: (a) the character of the murmur,



which is, as a rule, softer and less intense than that due to valvular lesions; and (b) the antecedent history of the patient. Thus, relative insufficiency of the mitral segments probably exists in patients in the middle period of life, and particularly in those in whom the previous history furnishes such etiologic factors as renal disease, syphilis, or alcoholism; or in persons who exhibit arterio-sclerosis or organic disease of the aortic valve and an apex-systolic murmur. On the other hand, if the signs of mitral regurgitation occur in a younger subject or in one who has been afflicted with acute or subacute rheumatism, it is hardly probable that the mitral-valve segments are the seat of chronic endocarditis of rheumatic origin. Again, if present in chronic renal disease, with concurrent symptoms of high arterial tension and of left ventricular hypertrophy—accentuation of the aortic second sound, a mitral systolic murmur—it is to be regarded as being due to relative insufficiency. I believe that a rare sequel of mitral incompetency is mitral stenosis, owing to the contraction of the auriculo-ventricular orifice, with, in some instances, progressive cohesion of the free edges of the leaflets from the base upward.

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### MITRAL STENOSIS.

**Definition.**—Constriction of the left auriculo-ventricular orifice, due either to a thickening or adhesion of the segments. With few exceptions adhesions of the free borders of the valve or of the chordæ tendineæ obtain.

**Special Pathology and Etiology.**—It is to be recollected that the constriction may be almost inappreciable, and yet that an uneven, roughened surface be presented, producing a murmur as the blood-stream enters the ventricle; on the other hand, a high degree of constriction may be encountered. Thus, in the *funnel-shaped* form of mitral stenosis the aperture may be so small as scarcely to admit the passage of a goose-quill. When moderate in degree the tip of the index finger is admissible; in the *button-hole* form the slit may be so narrow as not to allow an object larger than a shirt-button to pass through it. The *funnel* variety is common in children, and, in occasional instances, is possibly a congenital condition, while the button-hole variety is comparatively rare in childhood. In adults, however, the funnel-shaped constriction is rare, while the button-hole valve is quite common; in 62 *postmortem* examinations only 3 showed funnel-form contraction (Hayden and Fagge). Mitral stenosis is, as a rule, dependent upon a mild or limited endocarditis that is usually of rheumatic origin. It is more common in young adults and in children after the fifth year than in older persons, and it is more frequent in females than in males, for the reason that the affections that are causally related to endocarditis occur more frequently in the former sex (rheumatism, chorea, chlorosis). The endocarditis of measles and scarlatina may also lead to narrowing of the mitral orifice, and I quite agree with Osler in the belief that whooping-cough, owing to the great strain that it imposes upon the heart-valves, may be account-

able for certain cases. In adults arterio-sclerosis and chronic nephritis may induce fibroid changes in the mitral leaflets, with resulting stenosis.

**Mechanical Influence of the Lesion.**—On account of the obstruction of the blood-stream at the mitral orifice during diastole, the task of the left ventricle becomes greater than normal, and in consequence of this its walls hypertrophy. They may be found to be one-fourth or even one-half inch (1.2 cm.) in thickness, the normal thickness being only three-twentieths of an inch (3.7 mm.). Under these circumstances dilatation of the auricle comes on early, and in the later stages it may be extreme, the walls now becoming much thinner than in the normal heart. For a varying period of time the increased power of the heart due to hypertrophy of the left auricle and that due to an increased resistance to the circulation that is the result of the mitral lesion are exactly balanced. At a comparatively early period, however, the auricle can no longer maintain this equilibrium; and then, owing to retardation of the current from the pulmonary veins to the auricle, the vascular tension in the lungs and right ventricle is increased. The right ventricle, in seeking to overcome the obstruction, becomes greatly hypertrophied and dilated, and late in the disease tricuspid incompetency supervenes with its usual sequences. The hypertrophy of the latter chamber counterbalances the lesion during the greater part of the period of compensation. For a brief time the left ventricle exhibits no abnormal proportions. Later and at autopsies its cavity is found smaller and its walls thinner than the normal, these conditions being due to its abnormally light labor. The apex of the heart is formed almost exclusively by the greatly enlarged right ventricle. If the left ventricle be hypertrophied, it is owing to the existence of associated mitral incompetency.

**Symptoms.**—The subjective symptoms are scanty and of slight value in forming the diagnosis. During the period of full compensation there may be an entire absence of symptoms except on going up stairs or on attempting some unusual muscular effort, when dyspnea appears. The vegetations previously described are sometimes quite friable, and when so, may be swept from the valves into the circulation and give rise to the phenomena of cerebral embolism (aphasia and hemiplegia). The same conditions may arise, and in the same way, from recurring endocarditis, to which such patients are specially liable. The patient in well-marked cases presents an anemic appearance: a stitch-like pain in the apex-region is frequently present, and active exertion, by overtaxing the left auricle, induces cardiac palpitation and dyspnea.

*After failure of compensation* the symptoms referable to the pulmonary system are almost identical with those manifested in mitral incompetency. Owing to the pulmonary engorgement the dyspnea is constant, and is increased by over-exertion. After severe or prolonged physical exercise congestion, followed by edema of the lungs, may supervene, attended by a copious blood-stained, serous expectoration. True hemoptysis may arise from time to time. The increased tension in the pulmonary vessels being practically constant, sclerosis, followed by atheromatous degeneration of their walls, is a frequent occurrence, and may accidentally result in pulmonary apoplexy. Intercurrent febrile attacks (due usually to recurring endocarditis) are common, particularly in the later stages, and are attended with marked aggravation of the circulatory disturbances.

Among other things, mitral stenosis differs from mitral incompetency in that general anasarca is rare, though enlargement of the liver and other evidences of portal congestion (including ascites) are not wanting.

**Physical Signs.**—*Inspection.*—The apex-beat is not displaced unless there be excessive enlargement of the right ventricle or associated hypertrophy of the left. There is usually present a visible pulsation in the second left intercostal space, and sometimes in the third and fourth interspaces, occasioned by increased tension in the pulmonary artery; and there is also a diffuse impulse along the right border of the sternum. Epigastric pulsation is common. A prominence having its seat over the fifth and sixth left costal cartilages and the lower half of the sternum is observed, particularly in children. After failure of compensation the impulse is feeble and undulates, with engorgement and pulsation of the jugular veins.

*Palpation* discovers a presystolic thrill in a great proportion of cases. In certain instances active physical exertion may render this appreciable, or when in the recumbent posture on the left side the elevation of the arms may accomplish the same result. It is, however, absent in rare instances before failure of compensation occurs, and more frequently by far after the latter event. This fremitus is best felt over the third and fourth (less frequently the fifth) interspaces, just within the nipple, and during expiration. It commences after the second sound (during the diastole) as a purring fremitus, increasing steadily in volume and intensity, and terminates abruptly with the severe shock of the new impulse. The fremitus is pathognomonic, and may be relied upon in the absence of the murmur. The heart's impulse is most forcible over the lower portion of the sternum and along the right border, being due to the enlarged right ventricle; in a smaller proportion of cases, in the

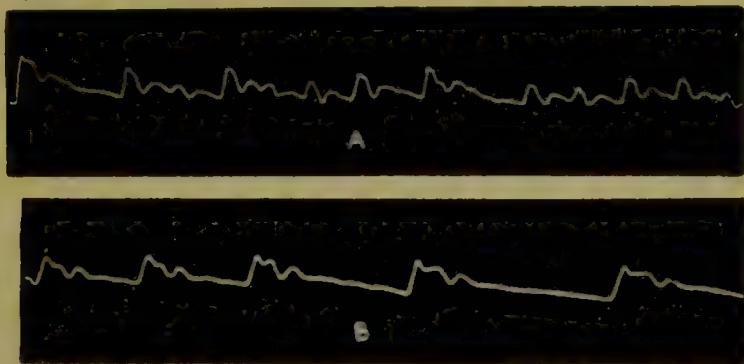


FIG. 53.—Sphygmograms in a case of mitral stenosis treated by extract of convallaria, and subsequently by digitalis: A, before treatment, showing the interpolated pulsations; B, after treatment (Sansom).

fourth and fifth interspaces to the left of the sternum. The radial pulse is small, compressible, and markedly irregular as the propulsive power of the right ventricle diminishes. The sphygmographic tracing is notably irregular (*vide* Fig. 53).

*Percussion* shows an extension of heart-dulness to the right, frequently 5 centimeters (2 inches) beyond the sternal margin, as a result of hypertrophy of the right ventricle, and upward as high as the sec-



ond rib on either side of the sternum. Increase in the cardiac dulness to the left also occurs not infrequently, and is attributable to excessive enlargement of the right ventricle, though more often of the left ventricle in consequence of associated mitral insufficiency.

*Auscultation* reveals a rough, presystolic murmur, which may be characterized as churning or rolling, acquiring increased intensity toward its termination. Its point of greatest pronounciation is just above and about one inch within the normal apex-beat. The area of transmission is generally quite limited, not exceeding a couple of inches in any direction. Griffith, however, has shown that the murmur is not seldom widely transmitted. This murmur sometimes exhibits atypical characters: it may be brief and low-toned, and may be audible on one occasion and then disappear for a considerable period. After the right ventricle becomes weak the murmur may lose its characteristic sudden termination, or may entirely absent itself either temporarily or permanently. In most cases the clear, accentuated first sound is retained, even though the murmur disappears. Improvement in the muscular power of the heart as the result of judicious treatment may cause the murmur to reappear, and I have seen such an occurrence in a case associated with mitral incompetency at the Philadelphia Hospital. For purposes of diagnosis nothing is so vitally important as the time and rhythm of the murmur, and in his examination the observer must therefore palpate the heart, and not the radial pulse, while practising auscultation. The finger as well as the ear will thus become sensible of the systolic shock which replaces the cardiac impulse, and it will be noted that the murmur terminates at the same moment. In cases in which the impulse cannot be felt the finger should be placed over one or other carotid, since here the pulse is practically synchronous with the systole of the ventricle. In the vast majority of the cases the murmur occupies only the latter half of the diastole, though occasionally it is sustained throughout the whole of the long pause. Owing to the presence of right ventricular hypertrophy the pulmonic second sound is greatly accentuated, being distinctly audible at the apex, while the aortic second sound is often absent or only feebly marked. Reduplication of the second sound is not rare, and is quite characteristic when it occurs.

*Secondary Murmurs.*—As previously pointed out, the murmur of mitral stenosis may succeed that of mitral incompetency, but this is comparatively rare. Neither does the mitral stenosis follow aortic valvular disease, save in the rarest instances, and in the vast majority of instances it is a primary affection. Secondary murmurs are not uncommon, however. Among these the bruit of *mitral incompetency* is relatively frequent. After compensation is ruptured the murmur of *tricuspid insufficiency* usually becomes audible at the lower end of the sternum, and persists until the end. At this period the presystolic murmur undergoes certain modifications, as already indicated.

**Diagnosis.**—The distinctive features of mitral stenosis are—(1) A presystolic thrill at the apex. (2) An increase in the precordial dulness upward and to the right. (3) A murmur which (*a*) has its seat above, yet near, the normal apex-beat; (*b*) is strictly localized; (*c*) is presystolic in time, terminating abruptly with the systolic shock (sharp

impulse); and (d) is "churning" in character. (4) A marked accentuation of the pulmonic second sound.

**Differential Diagnosis.**—When the murmur of mitral stenosis is very brief, it is difficult to eliminate a mere roughening. In the latter condition, however, there is no increase in intensity of the murmurs on exertion or when the arms are uplifted, and there is no right ventricular hypertrophy. From simple mitral stenosis the lesion of *mitral incompetency* is easily distinguished by its systolic rhythm, greater area of transmission, and by the soft, more flowing character of its murmur. A combination of the two lesions, however, is a more frequent occurrence than that of pure mitral stenosis; and under such circumstances it is with great difficulty that the two murmurs are separated. The presence of the systolic murmur is distinguishable by its synchronism with the impulse or carotid pulse, and by its area of transmission to the left as far as the axilla. If now the stethoscope be applied just above and to the right of the normal apex, a limited superficial area will be found where a presystolic murmur is distinctly heard. Points can also usually be found where one continuous bruit, covering a portion of the period of diastole and of systole, is audible. The presystolic murmur is sometimes, and especially after failure of compensation, entirely veiled by the systolic.

In *aortic regurgitation* the presence of a presystolic thrill and murmur has rarely been recorded, and Fisher, Phear, and others have called attention to their presence in adhesive pericarditis as well as in simple dilatation. When, as is usual, a purely diastolic murmur is also present in the aortic area, together with strong correlative evidence of aortic regurgitation, the diagnosis of mitral stenosis must be made with extreme caution.

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## TRICUSPID INCOMPETENCY.

(*Tricuspid Regurgitation.*)

**Definition.**—An imperfect closure of the tricuspid valve, due either to a dilatation of the right ventricle that is secondary to mitral or lung-disease, or, less frequently, to an inflammatory shortening of the valves.

**Pathology and Etiology.**—As a primary disease tricuspid incompetency is rare. It, however, is not uncommonly due to chronic organic changes, though originating in fetal endocarditis. After birth this variety is most common during childhood, and the frequency of occurrence is in inverse ratio to the age. At any period of life, however, chronic affections of the lungs or organic disease of the left side of the heart may, by augmenting the tension in the right ventricle, produce chronic interstitial changes in the tricuspid segments. These latter, however, are usually of mild grade. I have observed in autopsied cases of chronic bronchitis associated with emphysema, and in pulmonary tuberculosis, that the chief reason why extensive lesions of these valves are seen so rarely is to be found in the fact that dilatation of the right ventricle is soon followed by relative insufficiency, and thus the strain



is in great part removed from the valves themselves. And yet, according to the statistical studies of Byron Bramwell, the tricuspid valve is implicated in 50 per cent. of all cases of acute endocarditis, notwithstanding the rarity of sclerosis of these segments. He suggests that the acute form frequently results in cure because of the relatively diminished right intraventricular tension. In rare instances one of the leaflets has been ruptured by straining. The relative tricuspid insufficiency, produced in a manner analogous to mitral insufficiency, is an exceedingly common secondary condition in affections of the lungs and heart that cause hypertrophy and dilatation of the right ventricle (mitral incompetency and stenosis, emphysema, sclerosis of the lung).

*Secondary Alterations.*—In tricuspid leakage every systole of the right ventricle is accompanied by a reflux of venous blood through the imperfectly closed tricuspid orifice into the auricle, and thence into the veins. This causes venous stasis and gives rise to visible pulsation, and in this manner the engorged pulmonary circulation is relieved to some extent. A necessary unfavorable consequence, however, on account of the reflux current from the right ventricle, is the lessened blood-supply to the pulmonary arteries, even though the latter are found to be engorged. The already hypertrophied and dilated right heart now undergoes further enlargement in the same manner as in the hypertrophy of the left ventricle following mitral incompetency, though not to the same extent. In mitral incompetency the right ventricle compensates the mitral lesion after failure of the left auricle, but there can be no such effective compensatory reinforcement after failure of the right auricle in tricuspid incompetency, since the right heart is not reanimated by a fellow as is the left. The blood-stream flowing into the right ventricle during the period of diastole, however, is abnormally large, owing to moderately increased tension. When the right ventricle fails to maintain the pulmonary circulation, progressive dilatation of its chamber occurs, with a proportionate thinning of its walls until its dimensions are enormous.

**Symptoms.**—In most instances the indications of the primary or causal affection must be noted, though these are often more or less screened by the more characteristic features of the disease under consideration. The symptoms of tricuspid incompetency point to passive congestion of the lungs and engorgement of the systemic veins, and they have been described in connection with mitral lesions. *Cardiac dropsy* is common, though present in by no means all cases. Frederick Taylor<sup>1</sup> contends that *ascites* is absent frequently, because the liver acts as a diverticulum to accommodate the excess of venous blood.

**Physical Signs.**—*Inspection.*—Venous pulsation, caused by the backward blood-wave from the right ventricle at each systole, is a pathognomonic sign. It is confined to the lower portion of the jugular veins so long as the valve that lies above the jugularis remains closed, but soon this yields, and then the veins seem to pulsate through their entire course with each cardiac systole. This is best seen when the patient is in the semi-recumbent posture, and is more marked in the right than in the left side. The subclavian and axillary veins may also be seen to pulsate, but rarely. The veins appear to be everywhere en-

<sup>1</sup> *Lancet*, Nov. 22, 1890, p. 1126.



gorged, producing a cyanosis that is more noticeable if the breath be held when in full respiration. Tricuspid incompetency may be shown by pressing on the vein with the finger rather firmly, commencing just above the clavicle and passing upward, thus emptying it of blood. If, now, the right ventricle be capable of producing a return wave sufficiently powerful to overcome the valve in the external jugular, pulsation is seen to take place—also from below—in the vessel slowly and increasingly until the vein, as far as the point compressed, becomes filled. The vein fills “by jets synchronous with the heart-beat” (Sansom). Again, an impulse may be communicated to the jugulars from the underlying carotid artery; if this be the true explanation in any given case, the light pressure upon the vein below does not arrest the pulsation above, as is the case in tricuspid incompetency. Not rarely there is noticeable a feeble systolic venous pulse, due to the weaker contraction of the right auricle as compared with that of the right ventricle (*anadichrotic venous pulse*). The area and seat of the apex-beat vary with the nature of the positive affection: in mitral incompetency, for example, the beat is displaced to the left and downward, while in uncomplicated mitral stenosis no appreciable displacement occurs. To the right of the sternum an undulatory pulsation is seen, due to contraction of the right auricle and ventricle, but this is not characteristic, since it may take place in simple mitral stenosis without tricuspid regurgitation. Epigastric pulsation is almost invariably observed.

*Palpation* detects the heaving impulse of the right ventricle in the upper epigastric region. Rhythmic expansile pulsation of the veins of the liver is quite characteristic and is usually detectable. To obtain this sign the patient should lie on the back with the arms raised, and the examiner should place the palm of his left hand over the right mid-axillary region, and that of the right hand over the upper abdominal region. He will thus be enabled to feel an expansile pulsation of the liver synchronously with the ventricular systole. This is to be carefully distinguished from mere systolic depression of the organ due to the impulse of an enlarged right ventricle, transmitted through the diaphragm and left lobe of the liver to the epigastrium.

Popoff and others have also noted an inequality in the radial pulses in tricuspid regurgitation. This is probably due to the pressure of the enlarged auricle.

*Percussion*.—The extent and form of precordial dulness are variable according to the nature of the causal disease, but a dulness extending far beyond the right edge of the sternum is especially indicative of this lesion.

*Auscultation*.—A systolic murmur having its seat of greatest intensity at the base of the ensiform cartilage (*vide* Fig. 54) is almost constantly audible. It is clearly conveyed to the left one inch beyond the lower sternal margin, and to the right and upward for an equal distance beyond the limit of cardiac dulness. It is soft in character, short, and low-toned. Additional murmurs, due to primary lesions, are often heard, and usually at other orifices. The pulmonic second sound is accentuated.

**Diagnosis**.—I believe that the most valuable symptom for diagnosis is the venous pulse, whether observed clearly in the neck or de-

terminated positively by bimanual palpation of the liver, as before described. Either of these signs alone suffices. The murmur is generally audible, and when so is a most valuable aid to the diagnosis. The **differential diagnosis** between mitral and tricuspid regurgitation is easy when either exists alone, if it be remembered that the seat of greatest pronounciation, the area of transmission, and the character of the respective murmurs are widely different. But it is sometimes ex-

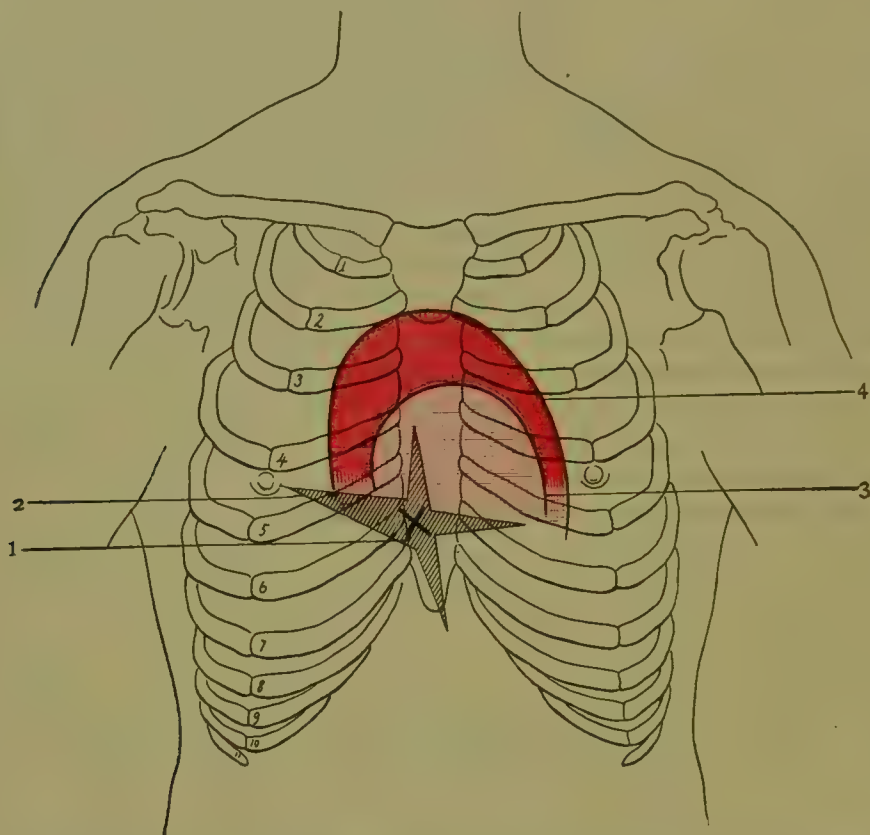


FIG. 54.—1, Seat of greatest pronounciation; 2, chief direction of conveyance; 3, boundary-line of absolute dulness; 4, boundary-line of relative dulness (modified from Sahli).

tremely difficult to discern positively a faint tricuspid murmur when it develops secondarily to the more pronounced murmur of mitral incompetency. If a careful observation of the murmur fails to establish the diagnosis of tricuspid insufficiency, as sometimes is the case, absolute reliance should, in my opinion, be placed upon the venous pulse when present, and the absence of the latter sign should exclude this disease.

### TRICUSPID STENOSIS.

THIS is a rare condition, occurring as a congenital and an acquired disease with about equal frequency. As a primary, independent disease tricuspid stenosis is very rare, being usually seen in association with organic disease of the left side of the heart. The lesions of mitral and tricuspid stenosis are observed to be combined most frequently,

while those of tricuspid stenosis and aortic insufficiency coexist less frequently. The morbid changes are practically identical with those of mitral stenosis, the right auricle becoming dilated, and this being followed by general venous stasis. The effect of tricuspid stenosis upon the right ventricle is the same as that of mitral stenosis upon the left ventricle. The right ventricle, however, is usually hypertrophied, owing to the obstruction in the pulmonary circulation that results from the combined valvular deficiencies.

**Special Etiology.**—The fact that mitral and tricuspid stenosis frequently have a common cause, acting concurrently, can scarcely be doubted in view of their frequent association and pathologic identity. Hence the statement that rheumatic antecedents are furnished by the history in from 30 to 40 per cent. of the cases of tricuspid stenosis need excite no surprise. As in mitral stenosis, so in tricuspid, sex is a potent factor, the statistics of Bedford, Fenwick, and of Leudet (which embrace a total of 160 cases) showing a ratio of 5 to 1 in favor of the female sex.

**Symptoms.**—The symptoms are those of the combined affections.

**Physical Signs.**—*Inspection* sometimes reveals a feeble venous pulse in the jugulars, due to right auricular systole, and hence presystolic in time. *Palpation* may detect a presystolic thrill over the body of the right ventricle. *Percussion* may enable the observer to indicate the enlarged right auricle. *Auscultation* gives usually an audible presystolic rolling murmur, which is best heard over the lower sternum and along its right border. The above physical signs are to be relied upon in *uncombined* cases, which are exceedingly rare. On the contrary, it is difficult in the extreme to differentiate the signs of tricuspid stenosis from those of the lesions with which it is almost uniformly associated—viz. mitral stenosis and aortic insufficiency. Hence a positive diagnosis of tricuspid stenosis is impossible save in the rarest cases.

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## PULMONARY INCOMPETENCY.

(*Pulmonary Regurgitation.*)

THIS is an exceedingly rare complaint, that results from acute (malignant) or chronic endocarditis after birth; it is also rarely due to a congenital malformation. In the latter form union of two of the segments is often observed; in the former, the usual sclerotic processes, with the occasional adhesion of one or more segments with the pulmonary artery wall, may be noted. The effect of the lesion is to cause hypertrophy and dilatation of the right ventricle. The *physical signs* furnish no diagnostic characteristics. There is developed a diastolic murmur which is most audible in the second pulmonary interspace, and is transmitted to the lower sternal region. This is indistinguishable from the murmur of aortic regurgitation. The water-hammer pulse and marked hypertrophied dilatation of the left ventricle are present in the latter complaint, however, and are absent in pulmonary regurgitation. In pulmonary insufficiency, on the other hand, hypertrophy and dilatation of the right ventricle ensue.



## PULMONARY STENOSIS.

A QUITE frequent form of congenital malformation of the heart is the narrowing of the pulmonary orifice. In the rarest cases it is of post-natal date, and may result in induration, contraction, and fusion of the segments. In one of Osler's cases the orifice "was only two millimeters in diameter, with vegetations of acute endocarditis on the segments." Even in cases occurring in after-life it is to be borne in mind that the etiologic factors are in all probability chiefly operative during fetal existence. I saw one case in which the pulmonary artery near the valve was contracted to one-half its normal caliber. Myocarditis with resulting contraction of the conus arteriosus may cause pulmonary stenosis, and some of the cases that originate during adolescence and later in life are due to atheromatous change, while others possibly are the result of chronic endocarditis or direct violence. Ulcerative endocarditis is occasionally responsible for the condition. The lesion is compensated by an hypertrophy of the right ventricle, following which dilatation and tricuspid incompetency may appear.

**Symptoms.**—Cyanosis and distention of the systemic veins are observed.

**Physical Signs.**—A systolic thrill may be felt at times over the base. There is considerable enlargement of the right ventricle, as elicited by percussion and palpation, and a systolic murmur of greatest distinctness is audible, as a rule, in the second left intercostal space near the sternum or at the junction of the third left costal cartilage with the sternum. It is harsh, superficial, and transmitted a short distance upward and to the left. Occasionally this murmur is heard best at the aortic valve, but it is never conveyed to the vessels of the neck, and hence is easily distinguished from the aortic systolic murmur. Its harsh character and loudness would serve to obviate confusion so far as functional or anemic murmurs are concerned. The pulmonic second sound is weak, and, not rarely, there is a diastolic murmur of the same character, indicating pulmonary regurgitation. Sansom holds that disease of the pulmonary artery (contrary to other forms of organic heart-disease) predisposes markedly to pulmonary tuberculosis. I have at present under my care a tuberculous patient in whom there is a double murmur audible at the pulmonary orifice.

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COMBINED FORMS OF CARDIAC DISEASES.

VARIOUS and frequent combinations of organic lesions occur, and it may be asserted safely that in more than one-half of all the cases combined murmurs are exhibited, and that a much higher percentage appears before the fatal termination. As I have already stated, stenosis of an orifice when due to valvular disease is associated with incompetency of the corresponding valve. Thus aortic stenosis is constantly combined with or followed by aortic incompetency, and in like manner mitral stenosis by mitral incompetency. The association may also have reference to lesions at two or more different valves, and according to the

elaborate table of F. J. Smith, the relative frequency of the chief murmurs found in combination is as follows :

Aortic diastolic and systolic and mitral systolic,	16.55 per cent.
Aortic stenosis and mitral stenosis,	6.12 "
Aortic diastolic and mitral systolic,	5.21 "
Aortic diastolic and systolic and mitral presystolic and systolic,	3.77 "

When two lesions coexist at the same valve, the one may compensate, in part at least, for the other, as, for example, in the case of aortic stenosis in association with aortic regurgitation. Here the stenotic deficiency lessens the reflux current from the aorta into the left ventricle during the diastole; hence the latter receives a correspondingly diminished amount of blood. During the contraction of the ventricle undue distention of the aorta is prevented, both on account of the narrowing at the aortic orifice and the relatively lessened contents of the hypertrophied ventricle. Similarly, in dominating mitral incompetency an associated mitral stenosis by lowering the strength of the regurgitant current renders the conditions more favorable. Relative insufficiency at the mitral valve, followed by aortic insufficiency, is most probably salutary in its effects, preventing, as it does, over-distention of the left ventricle, and also the over-filling of the arterial tree and the possible rupture of the blood-vessel. On the other hand, mitral incompetency is sometimes secondary to aortic stenosis; and when so the latter defect hastens the unfavorable tendencies in the former.

Relative tricuspid incompetency, secondary to mitral disease, usually results in the development of a serious impediment to the systemic venous circulation, and often heralds a speedy fatal issue. It is probable that in advanced mitral disease the occurrence of a slight leakage at the tricuspid valve may be the means of obviating disastrous consequences to the right ventricle in case of undue strain.

**Physical Signs.**—These are confusing, but a systematic analysis often leads to the correct inference. That one of the valvular lesions predominates over all others is a fact of paramount importance for the solution of these cases. The chief lesions can usually be determined by noting the seat, the area of transmission, and the character of the most pronounced murmur; and more important still is the correct timing of any murmur that may be audible. The secondary alterations in the heart frequently coincide with the predominating murmur, and it will therefore be an aid to the observer to recollect the familiar fact that mitral murmurs are often secondary to aortic, and that tricuspid murmurs point to the coexistence of mitral disease. Unquestionably, a single observation of these cases, however carefully made, is often profitless, whilst repeated observations may be productive of tangible results.

**Complications of Valvular Disease.**—Most of these have already been spoken of at sufficient length, but to restate them collectively in this connection may prove useful to the student and physician. They are—(1) acute endocarditis (including the ulcerative form); (2) acute pericarditis; (3) pleurisy; (4) pneumonia; (5) nephritis, followed by uremia; (6) local or general arterial sclerosis; (7) chronic gastric or intestinal catarrh with intercurrent acute attacks; (8) embolic



processes; (9) angina pectoris; (10) edema of the lungs; (11) hypochondria and melancholia; (12) rupture of the skin of the extremities in consequence of excessive edema, with erysipelatous inflammation; (13) synovitis, a not uncommon complication; fever, swelling of one or more of the joints, and pain are the usual symptoms. The muscles of the extremities may also be involved simultaneously. It is highly probable that these manifestations are to be regarded as being of a rheumatic nature, though they are also met with in ulcerative endocarditis. (14) Febrile paroxysms occur at varying intervals of time, and are due to a variety of causes, as rheumatism, acute endocarditis, and pericarditis. Ulcerative endocarditis may also occur and be attended with an irregular type of fever.

**Course and Duration.**—When valvular disease consists in rupture of a segment the course is brief and usually proves quickly fatal. Apart from these exceptional instances the duration is measured by months, or more often by years or even decades. Statements applicable to all cases cannot be made, however, owing to the wide differences in different cases. Among the circumstances affecting the duration I would mention in particular the patient's mode of life, the hygienic conditions under which he lives, his occupation, mental condition, and the severity of the morbid processes. Every experienced physician has doubtless met with a small class of cases that have terminated fatally in from six months to a year, having developed in that period all of the serious phenomena and complications of the more chronic forms of organic heart-disease. In the preponderating proportion of cases, however, the course is exceedingly slow, and often cases have existed many years before they have finally been recognized. In numerous instances the patient follows his usual avocation, which may even be laborious, for years, and without discomfort. In other instances symptoms, as dyspnea on exertion, are so slight as not to excite suspicion.

Facts such as these render it obvious that the period of compensation is long, though its exact limits are indeterminable. In 12 instances of chronic endocarditis that have developed under my observation (some having lasted ten or twelve years) only 3 have reached the stage of broken compensation. The progress after failure of compensation is more definitely known, since frequent opportunities for observation are afforded. At this time the cases also exhibit wide differences respecting their duration; in my own experience they have varied from two to three months to as many years (rarely even longer), depending much on the patient's mode of living. The course may be shortened by severe external injury, intercurrent acute illness (especially febrile disease), vicious habits, straining efforts, and the like.

**Prognosis.**—The detection of a cardiac murmur should not alone lead to a gloomy prognosis. Says Osler: "With the apex-beat in the normal situation and regular in rhythm, the auscultatory phenomena may be practically disregarded." Individual cases require separate and careful consideration. It is well not to advance positive assertions until all the circumstances that may influence the prognosis of any given instance have been well weighed. Observation of a case for some weeks and months enables the physician to speak with greater confidence and knowledge concerning the probable outcome; and hence



it is the part of prudence to delay giving a positive prognosis for a considerable length of time. Prior to the occurrence of disturbances of compensation the prognosis is measurably favorable. After this pivotal event the prognosis as to life becomes wholly unfavorable, though the end is not necessarily near at hand. Disturbances of compensation that are attended with marked arrhythmia, urgent dyspnea, and general dropsy may, under proper treatment, admit of even complete relief. Later on, however, restoration of the balance of forces becomes only partial, and finally the above-mentioned symptoms become more pronounced; Cheyne-Stokes' breathing may then develop, and, after a prolonged and distressing struggle for breath, the patient succumbs. Death may also occur suddenly from cardiac paralysis. Among ominous and yet common complications and intercurrent affections may be cited again extensive edema of the lungs, pneumonia, typhoid fever, embolic processes, ulcerative endocarditis, acute endocarditis, obstinate gastritis, and nephritis. On the contrary, favorable indications are sound general bodily condition, good external conditions (absence of poverty, hunger, etc.), strong and regular action of the heart, absence of arterio-sclerosis and of rheumatic antecedents, and correct habits of living. Age influences the prognosis to some extent. In children under ten years the lesions are usually somewhat more rapidly progressive than in adults, and the compensatory hypertrophy is developed with corresponding rapidity; hence the period of failing compensation is reached earlier. This may be said to be a broad general rule, and I have found that it is one to which there are many exceptions. Among other reasons for the more gloomy prospect when heart-disease occurs in young children are the following: the mitral valve is generally implicated, the liability to rheumatic interurrences is great, and children, unless carefully controlled, overtax at play the reserve cardiac power when indulging in running and other forms of exercise. After the twelfth year the prognosis becomes more favorable. Sex is also a modifying prognostic factor, women bearing valvular lesions better than men, apart from the influence of childbearing, though even this is an influence the significance of which has been greatly magnified by many writers. To explain the more favorable outlook in women we have two main facts—viz. a less laborious as well as a more quiet life, and a diminished liability to arterio-sclerosis and involvement of the coronary vessels. The particular valve involved has some influence on the prognosis.

*Aortic regurgitation* gives, on the whole, the most favorable prognosis, particularly in those cases that begin in early adult life, granting, of course, that the patient regulates wisely his manner of living. Under such circumstances the lesion may be compensated for many years or even decades. The increased vigor of the left ventricle as compared with the right is conducive to longevity in this disease. After failure of compensation, I admit, the prospect is not as hopeful in aortic regurgitation as in mitral regurgitation, since restoration of compensation is not as readily accomplished in the former as in the latter variety. In the lesion under consideration a chief danger arises from associated arterio-sclerosis—a rather frequent occurrence in advanced life—and from implication of the coronary arteries. Much depends upon the condition of the latter vessels. When their lumen is narrowed starva-

tion of the heart-muscle quickly ensues, followed by myositic degeneration. Blocking of one of the branches of the coronary artery is the most frequent cause of sudden death in this affection. In *aortic stenosis* equally favorable predictions are warrantable when the disease is uncomplicated.

*Mitral regurgitation*, when a primary lesion, is propitious, except in the very young, and not infrequently the progress of the morbid process is apparently arrested. In a considerable proportion of cases the disease does not materially shorten the life of the sufferer. In a larger percentage, however, there is special liability to a renewal of the causative affections (*e. g.* rheumatism) and to pulmonary conditions of serious import, producing exacerbations and permanent aggravations of the disease. The gravity of these intercurrent complaints is also increased by the existence of the cardiac lesion. Failure of compensation at once renders the prognosis decidedly unfavorable. In *mitral stenosis* compensation of the right heart fails somewhat earlier than in mitral insufficiency, and hence the accidents and conditions referable to the lung (diffuse pulmonary apoplexy, edema) are not so long delayed as in the latter disease: this is also true of the later, more serious manifestations. I have learned by experience that mitral stenosis is better borne by women than by men, and better during adolescence and early adult life than during more advanced years. The congenital forms are comparatively benign. It should not be forgotten that mitral stenosis causes sudden death more frequently than any other form of organic disease of the heart except aortic regurgitation. *Tricuspid incompetency*, whether secondary to disease of the lung or of the left side of the heart, is extremely grave. It is usually indicative of dilatation following hypertrophy of the right ventricle. The vigor of the ventricle, however, can be re-established, and sometimes repeatedly.

**Treatment.**—This falls naturally into three subdivisions: (1) prophylaxis; (2) management during the stage of compensation; (3) treatment of the stage of non-compensation.

(1) **Prophylaxis.**—It can scarcely be doubted, as shown by the statistics of Sibson, that complete rest and protection of the surface during an attack of acute articular rheumatism lessen the average percentage of cases in which acute endocarditis develops. When the latter complication occurs in acute rheumatism the patient should keep to his bed for some time after all rheumatic symptoms have disappeared (two to six weeks) or until the improvement in the cardiac condition has ceased absolutely. This precautionary measure will often lessen the extent of the ensuing chronic endocarditis, and also increase the proportion of perfect recoveries. Suitable dietetic and medicinal treatment must necessarily be combined. When the physician is cognizant of hereditary predisposition to organic heart-disease, or has to deal with the arthritic diathesis (gouty or rheumatic) or the alcoholic habit, he can frequently, by timely advice and hygienic suggestions, direct his patient to adopt measures that will obviate the occurrence of valvular disease. All persons predisposed by heredity or otherwise should be told of the probable effect of muscular strain, alcohol, and other exciting factors; too often, however, when he sees his patient for the first time the physician is confronted by an already incurable malady.



(2) **Management during the Stage of Compensation.**—Two main objects are to be accomplished: (a) The avoidance of every agency that tends to aggravate or maintain the lesion or lesions. Under this head the detection and removal of all causal factors is imperative. Thus, if the patient's avocation entails undue muscular effort, it must be abandoned; violent exercise, as running up flights of stairs, heavy lifting, or straining at stool, is also dangerous and must be prevented. If alcohol has been a factor, it must be discontinued; if syphilis, it must be treated specifically. If there be present a rheumatic or gouty taint of the system, it must be overcome as far as possible by special measures. The recognized causes of rheumatism, as fatigue and exposure, must be avoided, particularly if the patient be comparatively young. Mental excitement and strong mental effort injuriously affect the cardiac lesion; therefore tranquillity of mind should be insisted upon, though moderate and systematic mental exercise has no risks for the patient. In the case of children at school careful supervision of their studies as well as of their recreative exercises is essential. Fright and sudden emotion must be avoided if possible. The use of tea, coffee, and tobacco should be rigidly prohibited.

(b) The *diet* of the patient demands careful regulation. Only a very moderate amount of food, composed for the most part of readily digested albuminous articles (milk, eggs, the lighter forms of meats, and stewed fruits), is to be taken, since overloading the stomach will disturb the action of the heart; particularly is this true at night. The carbohydrates may be allowed only in limited quantities, since they are apt to decompose and form gases that distend the stomach and intestines. For the same reason the coarser and more indigestible food-stuffs should be avoided. Small meals at short intervals is a plan of feeding that I can highly commend. The amount of liquids taken should not exceed the actual requirements of the patient, inasmuch as over-filling of the blood-vessel system increases the work of the already overburdened cardiac forces. Alcoholic beverages should not be used as a rule; but if the patient has been moderate in the use of alcohol, and particularly if he be advanced in years, light wines may be allowed in moderate quantity to aid digestion. *Carefully regulated exercise* is beneficial, but it must be gentle, and should be taken out of doors. As before intimated, a good general muscular development is an aid of no mean value to the conservative powers of the heart. Oertel, with a view to assisting the compensatory forces of the heart, has recommended graduated physical exercise; he advises that patients be instructed first to ascend low elevations, and later mountains of a considerable height, the object being to bring about full compensation. Great caution is to be exercised by the physician, however, since this method has been found to be inapplicable to a large percentage of cases. Cardiac distress, palpitation, and dyspnea are complained of by this large group of patients if other than the gentlest forms of exercise be undertaken. With respect to exercise, then, the sensations and experiences of each patient must be consulted before the physician can advise judiciously. Woollens should be worn next to the skin during both the warm and the cold seasons. The skin should be kept clean by daily sponge baths, and if these be followed by friction of the surface, the bodily nutrition will be improved and the



liability to intercurrent attacks of bronchitis greatly lessened. The bowels should be moved each day, and usually the use of stewed fruits suffices to accomplish this end; if not, salines, Rochelle or Carlsbad salts, and the bitter waters (Friedrichshall, Hunyadi-Janos) must be brought into requisition. In winter a warm climate may prove advantageous, though long journeys are often illy borne, owing to the fatigue induced thereby. If, despite the measures above indicated, the patient becomes anemic or his nutrition is notably impaired, a suitable change of air,<sup>1</sup> or the use of quinin, mineral acids, arsenic, small doses of mercury, and cod-liver oil, is to be recommended. Digitalis should not be employed when compensation can be preserved in other ways.

(3) **Treatment of the Stage of Non-compensation.**—The chief object to be kept in view in this stage is the reinvigoration of the exhausted cardiac muscle, and thus to relieve the impeded circulation. Sudden death may, though rarely, occur from the blocking of a branch of the coronary artery or from acute dilatation. Failure of compensation, however, begins gradually as a rule, the condition often existing without marked or characteristic symptoms; but its early recognition is important from the stand-point of therapy. Increased dyspnea on exertion, and nocturnal seizures of shortness of breath and irregular action of the heart (arrhythmia), are among the earliest clinical features. The latter symptom may have been present before, and particularly during active exercise in mitral disease, but now it is more marked, and may be constant. The patient's nutrition often suffers, and he is pale and rather feeble. Absolute quiet, liberal feeding with suitable food, and iron may in a little while restore the impaired cardiac tone. If this treatment fails, by the end of a fortnight a small dose of digitalis should also be exhibited (5 minims—0.333—of the tincture three times daily); the latter should be promptly withdrawn upon the disappearance of the symptoms. Decided indications of lost compensation are marked dyspnea and arrhythmia; the canter rhythm; an irregular, small, compressible pulse; and cyanosis, with or without the presence of dropsy. To meet the latter serious condition we must have recourse to the following means: (a) *Absolute rest in bed.* This diminishes greatly the work of the heart, and thus enables it to regain largely its former vigor. Rest joined with careful yet liberal feeding and attention to the bowels will often restore disturbed compensation in from one to two weeks. In 3 cases recently treated at the Medico-Chirurgical Hospital this method succeeded admirably.

(b) *Cardiac stimulants and tonics.* Of these, when occasion demands, the most important is digitalis, and this may be tried in any case in which dilatation exists. By stimulating the pneumogastric, by increasing the blood-supply to the heart-muscle, by causing the systole to be more complete and the period of diastole to be lengthened, digitalis becomes an invaluable aid to the nutrition of the cardiac muscles. In addition, the heart contracts more regularly and the blood-pressure in the peripheral circulation is raised. As a result of the use of this drug the tissue-calls

<sup>1</sup> Observation and experience have confirmed my belief that sea-air during the warm season and high altitudes at all times are injurious in their effects in valvular disease of the heart.

upon the cardiac forces from the outlying portions of the body are satisfied and the reserve energies of the heart-muscles are maintained.

In mitral disease the influence of digitalis is most beneficial, the pulse becoming slower, of better tension, and more regular as a rule, while the urine increases in amount. The dropsy, when present, often disappears under its employ. In mitral incompetency its good effects are ascribable to the powerful contractions of the left ventricle, whereby the normal blood-stream from the ventricle to the aorta is greatly increased, while the regurgitant current is not proportionately increased, because of the fact that the mitral defect is minute as compared with the aortic. On the contrary, the patient's condition is occasionally aggravated by the drug, because "the leak is increased as much as the normal flow" (Hare). In mitral stenosis digitalis, by lengthening the period of diastole, allows time for the blood to pass from the auricle through the narrowed mitral orifice into the ventricle. Slight toxic effects may sometimes result from digitalis, the pulse becoming thread-like and irregular, the urine scanty, and, as pointed out by Broadbent in connection with mitral stenosis, there may be two heart-beats to one pulse.

In *aortic regurgitation* digitalis exercises as great, if not as wide, an influence as in mitral disease: the theoretic view, however, that by prolonging the diastole digitalis causes overfilling of the left ventricle rests on too slender a foundation to be regarded as a valid objection to its use. It may, however, produce excessive hypertrophy if used continuously for too long a period. Hence its effects should be carefully noted, and the drug promptly withheld should over-hypertrophy be engendered. In all forms of organic heart-disease, though most frequently in aortic regurgitation, nausea and vomiting sometimes follow the administration of digitalis: when this is the case it should be stopped and other cardiac stimulants substituted, or the dose reduced to the point of tolerance, when it may be continued if adequate to maintain a proper effect. When secondary dilatation comes on in aortic stenosis, digitalis is needed to increase left ventricular power. The dose is to be calculated according to the degree of existing dilatation. When tricuspid incompetency is secondary to disease of the left heart, striking results are obtained from the use of digitalis; but when it exists alone—*e. g.* following emphysema or cirrhosis of the lung—digitalis often fails. The cardiac contractions, if they have previously been irregular, may become somewhat more regular, but the precordial distress will often be increased, while the circulatory disturbance, as evidenced by the objective signs, will remain unrelieved. If dropsy be slight or absent, 10 minims (0.666) of the tincture or 2 to 3 drams (8.0–12.0) of the infusion, three or four times daily, will suffice. If symptoms of decidedly unfavorable import be present, including marked dropsy, the dose should then be larger (of the tincture, minims x to xv—0.666 to 0.999; of the infusion, ʒss—16.0—every two or three hours) for two or three days, when the dose must be diminished or given at longer intervals. Quantitative estimations of the urine should be made during the use of the drug, and if the effect be good the daily amount will often be greatly increased; if bad, there will be a diminution rather than an increase in the amount. Other favorable influences and disadvantages have already been adduced. There are not a few patients in whom the symptoms of commencing failure of



compensation recur as soon as the drug is discontinued. To such digitalis may be administered continuously or until toxic symptoms are manifested. I believe that the solid preparations (powder and extracts) can be taken for longer periods than the liquid forms without exciting untoward symptoms. This suggestion should be followed particularly in cases that are seen at long and irregular intervals of time. Evidences of fatty degeneration and atheroma are not contraindications to its use, but are signals for the observance of extreme caution. It should, however, be a rule never to be broken to discontinue the digitalis when the symptoms of disturbed circulation have vanished. When it fails of its effect or is not well borne, and when, as often happens, the arrhythmia is not favorably influenced by it, the physician is compelled to resort to other cardiac stimulants. These are numerous, and, whilst their good effects are not comparable to those of digitalis in every respect, some of them seem to meet certain indications that are not met by this drug. Among the more important are nitroglycerin, strophanthus, strychnin, cocain, spartein, and caffein. Nitroglycerin in small doses is at the same time a cardiac stimulant and an arterial relaxant, and hence is more often useful in aortic than in mitral valvular disease. In larger doses, when left ventricular hypertrophy is excessive, as may occur when general arteriosclerosis is associated with aortic regurgitation and also (though rarely) aortic stenosis, it is highly useful, widening the blood-paths, and causing less powerful contractions of the heart. Strophanthus should be employed in instances in which digitalis must be interrupted, since the action of these two remedies upon the heart-walls is very similar. The tincture is usually employed, the dose (varying with the indications of each case) being from 4 to 10 minims (0.266–0.666) every three or four hours, and in controlling the irregularity or intermittency of cardiac action it is sometimes better in its influence than digitalis. Many cases of marked arrhythmia will not yield to either when but one is given; and in such I have occasionally obtained good results from digitalis and strophanthus in combination. Caffein citrate is also a good cardiac stimulant, but is superior as a diuretic. It should be stated that, rarely, strophanthus, like digitalis, does harm rather than good, being sometimes badly borne by the stomach. Under these circumstances I have employed, both in hospital and private practice, the following combination:

R <sub>x</sub> . Caffein. citrat.,	ʒj (4.0);
Strychninæ sulphat.,	gr. $\frac{1}{3}$ (0.021);
Sparteïn. sulphat.,	gr. ij (0.129).
Ft. capsulæ No. xij.	

Sig. One every three or four hours.

The above prescription is not only a good heart-tonic and stimulant, but also an equally good diuretic. Spartein is a potent diuretic and heart-stimulant when employed in doses of gr.  $\frac{1}{6}$  to  $\frac{1}{4}$  (0.010–0.016) every four to six hours, and is especially serviceable in organic heart-affections when dropsy as a symptom and nephritis as a complication exist. Strychnin, when given hypodermically in full dose, gr.  $\frac{1}{30}$  to  $\frac{1}{15}$  (0.002–0.004), is the most efficient cardiac stimulant known to medical science. It should be employed in this manner in cases in which there is sudden failure of heart-power with the development of serious symptoms. Given in doses of



average size, *per oram*, its effects in chronic valvular disease are not very striking. Atropin may be advantageously combined with it.

When the indications are urgent and the above agents are not available, diffusible stimulants, as ether or ammonium, may be used until more suitable remedies can take effect. Cocain simulates strychnin in its action. The dose is gr.  $\frac{1}{4}$  (0.016) every four hours, and the drug may be given with digitalis in pill-form. Later, systemic tonics are often demanded by the anemia and other constitutional indications, and here iron and quinin should be joined with strychnin. Unquestionably, the value of iron in full doses as an aid to the completion of the work of restoring broken compensation has been and is still scarcely appreciated by the profession at large. When iron disagrees, arsenic may be given instead. In many cases of failure of compensation the restoration of the balance of the cardio-systemic circulation can be greatly assisted by withdrawing a portion of the blood-vessel contents; and in other instances the heart cannot be stimulated to regain adequate power until the overfilled venous system is depleted and the intracardiac pressure thus reduced. There are two ways in which to attain this end:

(a) *Venesection*.—When the right heart is over-distended, as shown by its very feeble efforts at contraction, and the whole venous system is intensely engorged, as shown by marked cyanosis and orthopnea, bleeding directly from a vein is not only warrantable, but often imperatively demanded in order to save life. From 16 to 30 ounces (473.0–887.0) may be removed safely, and the heart's action will almost immediately be observed to grow stronger and more regular, and the pulse fuller and of better tension. As before intimated, the form of dilatation of the right ventricle that follows emphysema is disinclined to yield to digitalis. In such instances, following the suggestion of Osler,<sup>1</sup> I have obtained brilliant results from free bleedings.

(b) *Depletion by purgation* affords less pronounced relief to the heart, though it is of the greatest value in cases in which a moderate grade of cyanosis and dropsy exists. As in the case of venesection, a feeble, irregular pulse is not a contraindication to the use of purgatives, since the latter remove directly a considerable portion of the heart's burden. The purgative to be used will vary with different cases. I select at the outset Rochelle or Epsom salts, employing them after the method of Matthew Hay—*i. e.* from 1 to 2 ounces (32.0–64.0) of Rochelle or 1 to 1½ ounces (32.0–48.0) of Epsom salts, in concentrated solution, to be given from a half to one hour before breakfast. Watery evacuations (three to six in number daily) usually follow the administration of the saline; but, unfortunately, one meets with many patients in whom it produces symptoms of marked catarrhal irritation. Next to salines, the most satisfactory results have been obtained from the use of claterium; I often combine this with podophyllin and belladonna. I have never seen good results from the use of mercurials when the object has been to procure venous depletion, but they are of service in dropsy, and particularly in ascites.

Schott of Nauheim has introduced a special plan of treatment that is applicable to most forms of valvular disease, simple dilatation, and nervous affections of the organ. Twenty-one baths are given in one month, drop-

<sup>1</sup> For illustrative cases from Prof. Osler's wards see article by Leutler, *Medical News*, July, 1891.

ping one every fifth, fourth, third, and second days. The water contains sodium chlorid, calcium chlorid, and carbon dioxid, and the temperature ranges from 82°–95° F. (27.7°–35° C.). The first bath lasts seven or eight minutes; the time is then gradually lengthened, the temperature lowered, and the carbon dioxid increased. After the bath the patient is rubbed and allowed to rest for an hour.

Artificial Nauheim baths are successfully employed in certain American hospitals at the present time. They are prepared as follows: Five pounds of sodium chlorid and eight ounces of calcium chlorid are dissolved in one half bath (30 gals.—114 liters), the temperature of the water being 95° F. (35° C.). In a few days the water is charged with carbon dioxid by adding sodium bicarbonate (1 lb.—453.6) and HCl ( $\frac{1}{2}$  lb.—226.8), the latter just before the bath is taken. The effects are to lower the pulse-rate, to decrease the size of the heart, to stimulate the nerves, and, indirectly, the cardiac nutrition. There is also a tendency toward improvement of the skin and an increase of the urine.

Gentle resistance exercises (consisting of all the more reasonable movements that a person naturally makes, and resisted by an attendant) form an important element of the treatment, since they tend to stimulate the muscles and nerves and propel the blood from the congested veins. The Nauheim treatment is not suitable in aortic regurgitation, aneurysm, or fatty degeneration of the heart.

**Individual symptoms** frequently become so conspicuous as to demand special treatment.

(1) *Dyspnea and Orthopnea*.—When these conditions are caused by engorgement of the pulmonary vessels, the cardiac stimulants above detailed usually afford relief. Frequently the patient cannot lie down, in which case a suitable bed-rest often gives immediate comfort and support. For the severe attacks of nocturnal dyspnea (amounting sometimes to orthopnea), particularly when accompanied by cardiac palpitation, the subjoined formula has proved itself of great benefit:

R<sub>y</sub>. Sodii bromidi, gr. xv (0.972);  
Tr. opii deod., ℥<sub>x-xv</sub> (0.666–0.999).—M.

Sig. To be taken in one dose at bed-time.

In the late stages of heart-disease morphin, given hypodermically, is to be preferred in combating this symptom, and is entirely free from the usual objections to the habitual use of the remedy. Its influence for good is inestimable. Dyspnea may also be produced by associated bronchitis, edema, emphysema, and hydrothorax—conditions that must be treated according to the customary rules. Frequent physical explorations of the chest should not be omitted. Hydrothorax demands aspiration, and this repeatedly in some instances.<sup>1</sup>

In valvular disease (particularly aortic), owing probably to coronary arterio-sclerosis, paroxysms of severe dyspnea (cardiac asthma) are apt to arise. These are best overcome by nitroglycerin in combination with sodium bromid at bed-time, to be repeated as needful.

(2) *Cough*.—Cough is common after failure of compensation, and is due to bronchitis resulting from stasis in the pulmonary vessels. In

<sup>1</sup> When the chambers of the heart are greatly dilated care must be exercised in inserting the aspirating needle, lest the left ventricle be entered.



mitral disease it may come on before the rupture occurs. Beyond the treatment directed to the causal condition (the cardiac failure) nothing is needed to relieve the cough. It should be remembered, however, that these subjects are very liable to suffer from catarrhal bronchitis due to cold, and that unless the condition be promptly controlled, the compensatory power of the heart will suffer.

(3) *Hemorrhage* may take place, and generally from the lungs, though it may also proceed from the nose, stomach, bowels, or uterus. In a recent case of double aortic and relative mitral insufficiency attended with marked dropsy, rather copious hemorrhages occurred from the bowel, but with apparent relief to the patient. The hemoptysis, which is a rather frequent accompaniment of mitral lesion, is rarely excessive, and is probably always beneficial. I would advise against active treatment unless the hemorrhage is actually copious in amount, and would apply this statement with equal force to hemorrhage from other mucous surfaces in connection with organic heart-affections.

(4) *Palpitation* may be due to different causes, the recognition of which in each case is important if we would institute appropriate treatment. At times undue hypertrophy maintains a constant throbbing and distress in the precordial region, the former being distinguished by the strength of the impulse and by the full, tense pulse at the wrist. Palpitation is best met by the use of the tincture of aconite, Mj-iv (0.066-0.266) every four hours. With the aconite I frequently associate the bromids with excellent effect. Unless the patient's discomfort is significant, however, this symptom does not call for active treatment. The administration of a saline purge not infrequently serves to quiet the heart. The patient may suffer from pure nervous palpitation, in which case the diet and the condition of the stomach must be carefully looked to, while for the throbbing the bromids of ammonium and sodium, together with preparations of valerian, are the most reliable.

(5) *Anginose Pains*.—These are seen in aortic incompetency accompanied by sclerotic vessels, and more especially in mitral stenosis. When dependent upon rigid blood-vessel walls nitroglycerin should be tried; if the attacks be severe, amyl nitrite by inhalation deserves a trial, and, this failing, morphin and atropin may be employed hypodermically. The latter measures, as a rule, promptly relieve the patient's suffering. Local measures alone are sometimes sufficient when the pain is only moderately intense, and the ice-bag or Leiter's coils may be tried. The sedative effect of a blister (4 by 6 in.—10 by 15 cm.) has more often proved effectual in my experience, though its use should be limited to patients whose general strength is not impaired to a great extent.

(6) *Pain* referred to the stomach, and less frequently to the abdomen also, occasionally assumes prominence and is relieved with great difficulty. It is dependent, in part at least, upon obstinate subacute gastritis, and I have quite recently seen an instance of the sort verified by autopsy. Among many drugs tested in this case, opium alone gave relief. Usually the pain results from gaseous distention of the stomach and bowels, and is not intense, a mild laxative frequently relieving the pain. Should this fail, however, trial should be made of the carminatives in combination with some antiseptic agent, as salol or creasote.

(7) *Gastric Symptoms*.—Soon after compensation is broken the ap-



pearance of mild symptoms of catarrh of the stomach may be said to be the rule, and these yield to simple measures in addition to the cardiac stimulants and laxatives already indicated. But there are not a few instances in which such symptoms as gastric distress and uneasiness, constant nausea with frequent vomiting, particularly after food, take on an aggravated form. This condition is scarcely amenable to treatment. Such patients cannot, as a rule, take digitalis or strophanthus by the mouth; they sometimes, however, do well on the capsules before adduced composed of strychnin, spartein, and caffein. When the latter cannot be borne I employ hypodermically digitalin and strychnin or caffein citrate, the latter being made soluble by the addition of sodium benzoate in solution. Cases of this class reach an early fatal termination, as a rule. The symptoms may be partly due to gastric catarrh and partly to uremic intoxication.

(8) *Nervous Symptoms*.—Insomnia and internal restlessness are almost constantly present at some period in the course of heart-disease, and notably in the more advanced stages. The restiveness is rendered more distressing on account of hideous dreams and cardiac palpitation on awaking. For these phenomena stimulation often answers a better purpose than sedation. Hoffman's anodyne (3j—4.0—well diluted), spirits of chloroform (℥xv—0.999), or ether (3ss—2.0), taken in whiskey (3j—32.0) are serviceable. The elixir of ammonium valerianate is also of value when given in full doses. When a hypnotic is required to afford sleep, I prefer sulfonal in combination with camphor monobromate or the following powder:

R <sub>x</sub> . Sulfonal,	gr. xv (0.972);
Sodii bromidi,	gr. xx (1.296).
M. et ft. chart No. 1.	

Sig. To be taken at 8 P. M.

In the later stages there is no objection to the use of morphin hypodermically. Headache due to uremia may frequently be a troublesome symptom in connection with sleeplessness, and in such cases morphin is the remedy *par excellence*; it is to be supplemented by free purgation and cardiac stimulants. Should the right heart be found flagging, venesection may be practised.

(9) *Dropsy*.—Among the symptoms requiring special treatment in advanced valvular disease dropsy easily assumes the lead. As above pointed out, rest with attention to the diet and state of the bowels will often restore defective compensation even when accompanied by a moderate degree of dropsy. In the severe grades of failure of the balancing forces the cardiac stimulants and purgatives before mentioned often suffice to remove the dropsy for a considerable period of time. Later, however, it becomes obstinate, and refuses to yield to any of the known methods of treatment. The therapeutic indications, so far as the symptom under consideration is concerned, are for the use of diuretics and purgatives. Diaphoretics, particularly the hot-air and vapor baths, are not to be thought of, since they tend to depress the already weakened heart. While describing the action of digitalis as a cardiac stimulant, incidental allusion was also made to its action as a diuretic. In view of the fact that it raises the blood-pressure in the peripheral vessels and capillaries by contracting their walls, and because of its stimulating

effect on the heart, digitalis in large doses becomes a most efficient diuretic in cardiac dropsy. When the renal secretion is not free under its use, or when for some good reason it cannot be taken, I have frequently found that a combination of strychnin, spartein, and caffein (*vide supra*) will excite free diuresis. Nitroglycerin may also be prescribed, especially in cases presenting evidences of advanced arterio-sclerosis. An unirritating yet highly effective diuretic mixture in these cases is the following:

R<sub>x</sub>. Potassii acetatis,                      ʒj (4.0);  
       Inf. digitalis,                        ʒij (64.0).—M.

Sig. ʒss (16.0) every three hours.

Purgatives are of the utmost value. Frequently, after a few copious watery evacuations as the result of the action of hydragogue cathartics, a free discharge of urine can be established, when before the latter event it has been impossible. Salines and elaterium, with podophyllin and belladonna, are agents that have been already recommended as purgatives (to deplete the venous system), and these should be first employed in the order named. Compound jalap powder may also be combined with the elaterium. A course of calomel, followed by salines until free catharsis is set up, is valuable from time to time. Mercury is especially applicable when the liver is much enlarged and ascites is a marked feature, or when the history of syphilitic infection is obtainable. It may be combined with cardiac stimulants and other diuretics as follows:

R<sub>x</sub>. Pulv. digitalis,  
       Pulv. scillæ,                      āā. gr. xij (0.777);  
       Hydrarg. mass.,                    gr. xxiv (1.555);  
       Ext. belladonnæ,                   gr. ss (0.0324).  
       M. et ft. pil. No. xij.

Sig. One every three or four hours.

When efforts at relieving the dropsy by means of medicines fail, then the most dependent parts of the body, or those most swollen, should be scarified under strict aseptic precautions. Fine silver trocars with rubber tubes attached (Southey's tubes) may be inserted and the liquid allowed to drain off in a gradual manner.

*Means to Prevent Recurrence of Broken Compensation.*—When the compensation has been successfully established, the after-treatment must be prosecuted with vigor for at least a year. The cause of the rupture of compensation is most probably fibroid and fatty degeneration of the cardiac muscle, and hence the mere restoration of the compensatory power of the heart does not imply a complete cure of the impaired muscular structure of that organ. Much can be done, however, to overcome the tendency to degeneration by the persistent use of certain tonic remedies, as iron, cod-liver oil, arsenic, and mercuric chlorid, the latter two in small doses. I have obtained excellent results from the use of the following prescription in these cases:

R<sub>x</sub>. Liq. arsenici chlor.,            ℥xlviij (3.186);  
       Tinct. ferri chlor.,            ʒss (16.0);  
       Hydrarg. bichloridi,           gr. ss (0.0324);  
       Glycerini,            q. s. ad fʒij (96.0).—M.

Sig. ʒj (4.0) after each meal, well diluted.

This preparation may be taken indefinitely with occasional brief interruptions. The patient should lead a very quiet life, and follow rigidly all hygienic rules that tend to prevent the production of valvular disease, and avoid whatever tends to aggravate in the slightest degree the lesions that may already exist. Appropriate diet, it should be emphasized, is not inferior to appropriate medication in its salutary effect. Should the faintest evidence of failure of the right ventricle manifest itself, the patient must be put to bed immediately, and the foregoing treatment is to be carried out. I am of the opinion that the plan herein advocated not only renders the course of recurring attacks of failing compensation milder, but that, in a considerable proportion of the cases, the much-dreaded recurrence is thus prevented.

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### CARDIAC THROMBOSIS.

**Pathology.**—True cardiac thrombi are seen most frequently on the right side of the heart, in the auricular appendices, and, less commonly, in the right ventricle near the apex. They are of firm consistence, and are tightly adherent to the endocardium, considerable force being required to dislodge them. The color, while generally grayish-brown or red, varies with the age of the thrombus, being more colorless as it becomes older. Cardiac thrombi may be pedunculated or sessile, and their contour is, as a rule, more or less rounded. Recklinghausen and others have observed globular masses, the so-called “ball-thrombi,” in the auricles, without the slightest endocardial attachment. They vary greatly in size, from a mustard-seed to a hen’s egg, and sometimes exhibit calcareous degeneration. Cardiac thrombi may occur singly or in groups of considerable numbers. From the cavity in which they have their primary seat they may project into other chambers of the heart, or from the left ventricle into the aorta for a considerable distance. It is evident that fragments detached by the blood-stream from these cardiac blood-concretions will tend to lodge in various viscera and in the peripheral tissues, and set up embolic processes. Examined microscopically, degenerate round cells and detritus are revealed, but pus-cells are not seen. Secondary degenerative changes, and later softening, may take place in the central portions of a thrombus, and these areas may contain a reddish-brown liquid.

**Etiology.**—The causes of cardiac thrombosis are to be found chiefly in some previously diseased or injured condition of the endocardium, though sometimes alterations of the blood constitute a factor of considerable importance. The condition may occur in the course of both acute and chronic diseases, in which the intracardiac conditions favor the formation of a blood-clot. Hence it is seen in connection with organic diseases of the heart in which the valvular and often the mural endocardium are roughened, and the obstructive and regurgitant lesions at the various valves cause retardation in the blood-current. Chronic obstruction in the lungs may contribute to the result by slowing the circulation in the heart. Cardiac thrombosis has been observed in many



of the acute affections, and almost invariably there is a loss of endocardium, due to inflammatory action (endocarditis) at some point in the cavities of the heart. This becomes the seat of the fibrinous deposit which is subsequently imperfectly organized. Among the most important of these acute primary diseases are rheumatism, diphtheria, lobar pneumonia, and pyemic and puerperal conditions. It may be questioned whether, given a healthy endocardium, as contended by some writers, slowing of the circulation alone suffices to cause true cardiac thrombi.

**Symptoms.**—These will depend very much upon the rapidity with which the thrombus is formed, as well as upon its seat and dimensions. Thrombi invariably lack definiteness, and, as their effects are largely mechanical, signs of obstruction to the cardiac circulation and failure (more or less gradual) of the cardiac muscle are developed. The pulse becomes weak, rapid, and irregular; dyspnea, vertigo, and attacks of syncope are frequent; and later cyanosis may appear. It is probable that at times the liquefied products of a clot may be absorbed, producing blood-poisoning. When the thrombus is formed rapidly, all of the symptoms enumerated are suddenly developed and quickly assume a most serious phase. Rarely a valvular orifice, an efferent vessel, or the coronary artery may become blocked and instant death follow. Since the right heart is the most frequent seat for these thrombi, pulmonary embolism, attended with its usual symptoms, is a common event. When portions of a clot are broken off and swept into the systemic circulation, the clinical phenomena of cerebral, splenic, or renal embolism are exhibited.

The **physical signs** consist of a feeble impulse with marked arrhythmia; the area of dulness is somewhat increased to the right, and often upward; and the heart-sounds are greatly enfeebled and quite irregular, with marked change in any murmurs that may previously have been audible. In this condition a systolic pulmonary murmur may rarely be engendered.

**Differential Diagnosis.**—It is important to distinguish true cardiac thrombi, such as are above described, from the less dense and usually darker clots that are formed either immediately before or after death. The latter may seldom show an attempt at a very low grade of organization, and may present a somewhat decolorized appearance, but they do not adhere firmly to the endocardium. Moreover, *antemortem* and *postmortem* clots, as the latter may be appropriately termed, have a different causation from true thrombi. For instance, they are apt to form in diseases in which the fibrin-factors of the blood are greatly increased, as in pneumonia. Perhaps a more potent causal element is the progressive weakening of the heart-muscle, resulting in partial expulsion of the contents of the right ventricle; the blood that remains in the chamber is merely whipped up, and the deposition of its fibrin must thus be greatly favored. Such heart-clots may be generated if the endocardium be healthy, and cannot be separated positively from true cardiac thrombi by clinical observation.

The **prognosis** is uniformly bad and sudden death may be expected.

**Treatment.**—Beyond measures calculated to meet the symptomatic indications nothing can be suggested.

## HYPERTROPHY OF THE HEART.

*(Hypertrophia Cordis.)*

**Definition.**—Hypertrophy is an increase in the muscular structure of the heart, evidenced usually by an increased thickness of its walls. It is almost invariably associated with dilatation of the chambers.

**Pathology.**—When the two processes—hypertrophy and dilatation—coexist, they cause great enlargement of the organ. To this condition the term “*eccentric hypertrophy*” has been given. Hypertrophy without dilatation receives the name “*simple hypertrophy*,” and hypertrophy with diminution in the size of the cavities was formerly described as “*concentric hypertrophy*,” but this term should now be regarded as obsolete, inasmuch as the abnormally small chamber is known to be due to postmortem contraction of a normal or hypertrophied ventricle.

The increase in size may affect either the whole heart, one chamber on either side, one whole side, or but a single cavity (*general* and *partial hypertrophy*). The process may also be limited to a minute division of the heart (*circumscribed hypertrophy*). Owing to its important physiologic function the left ventricle is more frequently enlarged than the right, and though the auricles are not as often the seat of hypertrophy as the ventricles, the right auricle is more frequently involved than the left. The *weight* of the normal heart in a man of average size is approximately 9 ounces (255.0); in a woman it is 8 ounces (226.0). In bilateral hypertrophy, however, the weight of the heart may be greatly increased, though wide variations are exhibited; hearts weighing from 15 to 25 ounces (425.0–710.0) are seen in moderate grades of hypertrophy, and those of from 40 to 50 ounces (1134.0–1420.0) in extreme cases (*cor bovinum*). Measurements showing the thickness of the walls also indicate the degree of hypertrophy<sup>1</sup> and the exact seat of the enlargement when not general. Normally, the diameter of the left ventricle is from 8 to 12 mm. ( $\frac{1}{3}$ – $\frac{1}{2}$  in.); that of the right ventricle, from 5 to 7 mm. ( $\frac{1}{5}$ – $\frac{1}{4}$  in.); that of the left auricle, about 3 ( $\frac{1}{8}$  in.), and of the right, 2 mm. ( $\frac{1}{12}$  in.). Suffice it to state in this connection that under conditions of cardiac hypertrophy the normal thickness of the various cavity-walls is usually doubled, not infrequently trebled, and, rarely, even quadrupled. It must be noted, moreover, that in cases in which there is a concomitant dilatation the walls may appear thinner than is normal, while the measurement will show them to be in reality thickened.

The *shape* of the heart is also altered according to the seat and extent of the hypertrophy. If both ventricles are enlarged, the apex is widened and appears flattened; if only the left ventricle is involved, the apex is lengthened and is more or less pear-shaped; and if the right ventricle alone is hypertrophied (as in pure mitral stenosis), it may form the largest part of the apex, but the latter will be less conical than in health.

The papillary muscles and columnæ carneæ are greatly thickened, and, particularly in the eccentric form of hypertrophy, they are often decidedly flattened. In this form the septum frequently shows increased

<sup>1</sup> Measurements should not be attempted until the *rigor mortis* has been overcome by soaking the organ in water.



thickness—a condition that I have never observed in simple hypertrophy. The muscular trabeculæ generally assume greater prominence on the right than on the left side. The muscular structure is usually of a deeper red color and also firmer than normally. The hypertrophied left ventricle can, as a rule, be lacerated readily, while the right, as first pointed out by Rokitansky, may be tough and leathery. As the heart continues to enlarge it sinks lower in the chest-cavity; this is not, however, owing to an increase in size alone, but more particularly to an increase in weight. In hypertrophy of the heart there is a multiplication of muscular fibers, to which alone the enlargement of its walls is attributable.

**Etiology.**—Hypertrophy of the left ventricle (sometimes termed *general hypertrophy*) results from obstructions to the arterial circulation of whatever sort. These may be classified, according to their seat, into—(1) **Lesions of the Heart.**—(a) Aortic incompetency and aortic stenosis; (b) Mitral insufficiency; (c) The fibroid form of myocarditis; (d) Pericardial adhesions, particularly in the young. Late in life the heart may become atrophied. In such cases the rhythmic play of the ventricle is impeded, the adherent pericardium exerts a counter-traction force during the systole, and thus the work is increased beyond the capacity of the normal heart: in order to maintain a proper circulation, therefore, the muscle hypertrophies. There is no obstruction either at the orifices or in the arterial tree. In fibrous myocarditis a portion of the muscular tissue is more or less functionless, though compensated for by other healthier portions, while the rhythm of the heart is also greatly disturbed. In valvular disease the augmented tension in the ventricle induces the hypertrophy.

(2) **Abnormal Conditions of the Blood-vessels.**—(a) Narrowing of the aorta—*e. g.* congenital stenosis, external pressure, and the development of an aneurysm; (b) General arterio-sclerosis, by raising the pressure; (c) Increased arterial pressure, due to the contraction of the peripheral vessels in consequence of the local action of certain chemical and biologic irritants (lead, Bright's disease, gout, syphilis). In all of these cases, whether the blood-pressure is raised in larger or smaller vessels, increased cardiac action is essential to meet the demands of the system-circulation.

Attention should be called to the causes of the so-called "**primary idiopathic hypertrophy**," in which variety the above-mentioned etiologic factors are absent. The main causal conditions are—(1) Prolonged physical exertion, such as is necessary in certain occupations (blacksmiths, locksmiths, draymen, and athletes). (2) Constant over-distention of blood-vessels, as in the case of excessive beer-drinkers (*beer-heart*). Here the direct action of the alcohol upon the heart-muscle must also be taken into account. (3) Functional disturbances (neuroses), constant over-action of the heart, and even paroxysmal tachycardia—conditions excited by excessive mental excitement or emotion—tea, coffee, and alcohol may give rise to primary and general hypertrophy. In the latter category of cases it is the excessively rapid action of the heart that produces the hypertrophy.

**Hypertrophy of the right ventricle** develops secondarily to any condition that offers obstruction to the pulmonary circulation or to the blood-



current through the right ventricle. Among them may be mentioned—(1) mitral incompetency and stenosis; (2) emphysema, or cirrhosis of the lung (producing compression or obliteration of the vessels); (3) right-sided valvular lesions, particularly obstruction at the pulmonary orifice; (4) it is doubtful whether, on account of the normal situation of the right ventricle, pericardial adhesions induce hypertrophy of this chamber, as is sometimes claimed.

**Hypertrophy of the Auricles.**—Hypertrophy with dominant dilatation of the left auricle occurs in mitral disease, and especially in mitral stenosis. The right auricle hypertrophies, though not invariably, when the blood-pressure in the pulmonary vessels is pronounced from any cause. Stenosis of the tricuspid orifice is occasionally the sole cause of thickening of the right auricular wall, which also becomes hypertrophied in tricuspid incompetency.

**Symptoms.**—There may be an entire absence of subjective symptoms, since hypertrophy of the heart may be said, with rare exceptions, to be protective in character. When present, their intensity varies with the degree of the hypertrophy, which is then pronounced, as a rule, and often already attended by incipient dilatation. They may be *local* entirely, though frequently *general* as well. Of the former, precordial fulness and uneasiness are the most conspicuous. They are usually most annoying when the patient is in the recumbent posture, particularly on the left side, and when the hypertrophy is dependent upon nervous causes. Pain and palpitation are seldom complained of except by neurasthenics and patients suffering from enlargement due to tobacco and excessive muscular exertion. Decided aggravations of the local manifestations may follow the operation of influences that create a demand for increased cardiac action, such as undue mental emotion or excitement, physical exhaustion, active bodily exercise, and gormandizing.

The *general symptoms*, when present, may fluctuate or even intermit. Those most frequently observed are fulness in the head, often amounting to actual headache, tinnitus aurium, carotid pulsations, flushing of the face, flashing of light before the eyes, and often prominent eyeballs. These symptoms are attributable to the increased vigor of the cerebral circulation.

**Remote Effects.**—General or total hypertrophy promotes abnormal fulness of, and increased tension throughout, the arterial tree. Endarteritis and arterio-sclerosis are, as a consequence, frequent consentaneous developments in advanced cases, especially when the cause of the enlargement has been increased tension in the peripheral vessels, as in Bright's disease. With a circulation too forcibly carried on, as in hypertrophy, the sclerotic vessels are overstrained, especially the large ones near the heart, and are apt to rupture. The break often occurs in the brain (apoplexy) or in the lung (pulmonary apoplexy), and hemorrhage from the lungs (hemoptysis), due to left ventricular hypertrophy, is of greater frequency, I believe, than most writers are ready to admit.

**Physical Signs in Left-sided Hypertrophy.**—*Inspection.*—In females and in young children with yielding ribs there is seen precordial bulging. The intercostal spaces are much broadened and the visible apex-beat covers a greatly increased area, the extension being downward and to the left. The whole body of the patient, and even the bed on which he may be lying, may share visibly in the cardiac impulse.

*Palpation.*—In pronounced grades the impulse may be felt as low down as the seventh interspace and as far to the left as the axilla. In simple hypertrophy it is carried downward to the sixth intercostal space and outward to a point near the anterior axillary line. The impulse is slow, forcible, and heaving, lifting the fingers of the examiner at each systole. In eccentric hypertrophy (hypertrophy with dilatation), though heaving and forcible, it is somewhat more abrupt, and, partakes of the nature of the impulse in cardiac dilatation. Over the aortic orifice or second interspace, to the right of the sternum, a short diastolic impulse may also be felt occasionally (double impulse). The pulse in pure hypertrophy is full, strong, regular, and of normal rate; it is also prolonged, owing to increased tension. In eccentric hypertrophy it is soft, full, and somewhat accelerated.

*Percussion.*—The area of cardiac dulness is enlarged both in its vertical and transverse diameters. Traced upward, dulness may terminate in the second interspace, whilst to the left it may extend 1 or 2 inches (2.5–5 cm.) beyond the mid-clavicular line. When hypertrophy is of moderate extent, the left limit of dulness corresponds with the results of palpation and inspection; but when it is of immoderate extent, the extension of dulness does not keep pace with the systolic impulse, which is diffused to points beyond the limits of contact of the heart with the thoracic wall. If concomitant hypertrophy of the right ventricle be present, dulness will also extend to the right for 1 inch (2.5 cm.) or more beyond the right edge of the sternum.

*Auscultation.*—The sounds vary with the grade of the morbid process and the variety. In simple hypertrophy of marked type a prolongation of the first sound is always appreciable, and usually it is duller than the normal. The second sound is intensified, clear, and often ringing. The degree of accentuation depends partly upon the vigor of the left ventricle, though chiefly upon the condition of the blood-vessels. Reduplication of the second sound, due to high tension, is common (*e. g.* in Bright's disease). In dilated hypertrophy the first sound is clearer and more abrupt, while the second sound is less marked or even faint. Modification of the sounds just described occurs when hypertrophy is dependent upon chronic valvular disease.

**Hypertrophy of the Right Ventricle.**—One or more of the causal factors that produce augmented tension in the pulmonary vessels are present, and, if properly appreciated, will throw light upon the condition. There may be an absence of all symptoms if the hypertrophy exactly balances the result of the obstructive forces, and this state may be maintained for a long period of time. Undue exertion, however, soon leads to temporary dyspnea in many cases. When secondary to emphysema or cirrhosis of the lung the symptoms occasioned by the latter diseases, such as cough and dyspnea, may completely veil any symptoms that may be due to the hypertrophy. Discomfort in the cardiac region should, however, arouse suspicions of the existence of the latter condition. When dilatation of the ventricle supervenes, as is usual, and the clinical evidences of tricuspid incompetency develop, then pulmonary symptoms, due to venous congestion, are prominent; these are bronchial catarrh, shortness of breath, and the like. Later, general cyanosis and edema appear. As pointed out in the discussion of Mitral



Stenosis with permanently heightened tension and overgrowth of the right ventricle, the lung-vessels become atheromatous and the lung-tissue the seat of brown induration. Owing to the fact that the sclerotic vessels are easily ruptured, hemoptysis—a not uncommon event after sudden great exertion—is to be expected; intense pulmonary congestion and apoplexy may also be met with in hypertrophy with dominant dilatation.

**Physical Signs.**—These have been in the main detailed in speaking of affections of the mitral valve. Inspection discloses bulging of the sixth and seventh left costal cartilages and of the lower sternum. In the angle between the ensiform cartilage and the seventh rib an epigastric impulse may be visible, but more commonly the impulse is in the sixth interspace, close to the left edge of the sternum. It is also very generally seen to the right of the sternum, in the third and fourth interspaces, and particularly is this the case in eccentric hypertrophy, forming a highly characteristic sign. The apex-beat is therefore diffuse, the radial pulse is small, and in dilated hypertrophy it is increased in frequency, and is small and irregular.

*Percussion* shows the extension of cardiac dulness to a point an inch (2.5 cm.) or more beyond the right sternal border. When there is great increase transversely, dilatation is most probably associated and may predominate over hypertrophy. The *auscultatory* signs are not distinctive unless dilatation also exists, when the first sounds are clear and sharp. In simple hypertrophy the first sound is slightly prolonged and lower than in health. Owing to the high vascular tension throughout the lungs the second sound at the pulmonary valve is accentuated, and reduplication of the second sound may occur for the same reason.

It must be kept in remembrance that when advanced emphysema is present all the physical signs will be greatly modified, and may even be entirely negative, though the heart be of large size. Under these circumstances venous pulsation in the neck would be diagnostic of dilated hypertrophy of the right ventricle.

**Hypertrophy of the Left Auricle.**—This may be assumed to occur in mitral stenosis and incompetency in order to compensate for these lesions: it cannot, however, be recognized positively by physical signs. When the chamber is at the same time extensively dilated, the dulness may be extended upward to the left of the sternum, passing over the third and even second interspaces. At this point—the second interspace—a presystolic wave may now be noticeable.

**Hypertrophy of the right auricle**, associated with dilatation, is perhaps more common than its counterpart on the left side. It is secondary to tricuspid incompetency (rarely stenosis) and enlargement of the right ventricle, and hence has the same etiology as the latter conditions.

The *physical signs* are—systolic jugular-pulsation, sometimes a presystolic wavy pulsation over the third and fourth interspaces to the right of the sternum, extension of cardiac dulness to the same interspaces, and other signs of tricuspid regurgitation.

**Diagnosis.**—The recognition of cardiac hypertrophy is possible only by attention to the physical signs. Next to these, in point of diagnostic value, come the causes, which should therefore be diligently searched for; the rational symptoms are least in value, though usually



corroborative. It is difficult to establish a diagnosis, even approximately, when extensive emphysema coexists. As before pointed out, venous pulsation in the neck would point indisputably to right ventricular hypertrophy.

**Differential Diagnosis.**—Conditions that cause an increase in the precordial area of dulness, except hypertrophy, must be eliminated. (1) *Pericardial Effusion*.—A careful analysis of the physical signs and the history will suffice. (2) *Aneurysm*.—In this affection the enlargement is altogether upward and to the left or right. This fact, joined with the other evidences of aneurysm, should obviate error. (3) *Mediastinal growths* also enlarge the dull space mainly upward and to the right or left, though the point of cardiac contact may be increased and the heart carried forward. (4) *Displacement of the heart* does not give a heaving impulse nor an increased area of dulness; moreover, it usually furnishes its special cause (pleural effusion). (5) Abnormally narrow-chested persons present a considerably increased superficial zone of dulness, partly owing to the position assumed by the lungs and partly (perhaps chiefly) to their imperfect development. Since there is usually an entire absence of all other physical signs of hypertrophy, ordinary caution will exclude the latter complaint. (6) *Affections of the Lungs and Pleuræ*.—Left-sided pleurisy with retraction may, by exposing a large part of the anterior surface of the heart, give rise to signs of moderate hypertrophy. The presence of the former condition, the lack of expansion on deep inspiration, the displacement of the heart to the left and upward, and an absence of the causes of the latter should lead to a correct conclusion. (7) *Phthisis and cirrhosis of the lung*, with or without pleurisy, may in like manner produce apparent enlargement of the heart. It must also be remembered that cirrhosis of the lung is one of the causes of right-sided hypertrophy, and that the latter condition may therefore be present.

**Prognosis and Course.**—The course that will be pursued depends largely upon the stage at which the case has arrived and the character of its special cause. I have repeatedly found *postmortem* evidence of a moderate grade of hypertrophy in persons who died of other affections, and with especial relative frequency in those who had constantly followed manual pursuits. Simple cardiac hypertrophy, being compensatory as a rule, exerts in nearly all instances a salutary influence, and if the processes that constitute the causal factors are not steadily progressive, life may not only not be curtailed, but be greatly lengthened by its existence. Even in organic valvular disease of the heart hypertrophy prolongs life by overcoming the ill effects of the valve-lesion and by maintaining the normal circulatory equilibrium. But since in this class of cases the lesion is progressive despite treatment, a limit is reached sooner or later beyond which the increased vigor on the part of the heart cannot be maintained. The nutritive functions become inadequate in obedience to a natural law, and muscular degenerations then occur, followed by disturbances of the circulation due to cardiac weakness and secondary dilatation. It must, however, be recollected that the heart may at no time, in the course of certain cases, fully compensate for the causal condition—*e. g.* as when a valve ruptures with startling suddenness. Failure of the cardiac nutrition at once renders the

prognosis unfavorable. Serious symptoms, widely distributed, that have been previously mentioned as characterizing broken compensation, are certain to arise and gradually prove fatal, though, as a rule, temporary restoration of the cardio-circulatory system is obtainable. Occasionally, as the result of undue muscular exercise, acute dilatation, followed by a speedy termination of life, is observed. I believe that hypertrophy of the left ventricle warrants a more favorable prediction than can be made in hypertrophy of the right, and this for two reasons: first, the increased capacity for work of the left ventricle; second, the milder character of the many factors that are productive of left ventricular hypertrophy, as compared with those of the right. In special instances, however, the reverse may obtain, as when left-sided hypertrophy is associated with or caused by general arterial degeneration. It may be of advantage to the student and junior physician to recapitulate here a few of the chief points that are prognostically favorable as well as those that are unfavorable: **Favorable Conditions.**—(1) When the hypertrophic development fully compensates the causal lesion; (2) when the causes are removable or more or less amenable to treatment; (3) when the external conditions under which the patient lives, his habits, and general nutrition are good. **Unfavorable.**—(1) When signs of imperfect nutrition of the heart arise; (2) when evidences of advancing cardiac dilatation (dyspnea, rapid, irregular pulse, edema) show themselves; (3) when poverty, poor food, intemperate habits, and an unhygienic environment are all combined; (4) when apparent cardiac vigor suddenly gives place to dilatation and great cardiac weakness.

The **treatment** has for its prime objects the establishment of full, and the prevention of failure of, compensation (*vide* Chronic Valvular Disease).

*Over-hypertrophy*, as indicated by certain cerebral and thoracic symptoms, may result from the exercise of improper notions respecting the treatment of the causative lesions and of organic disease of the heart in particular. It requires careful dietetic and hygienic management rather than therapeutic activity. Briefly, the *diet* should be nutritious, but the more concentrated forms of food should be used very sparingly, and the daily quantity should be slightly less than that required in health. It must be non-stimulating, and tea, coffee, alcohol in all forms, and smoking must be prohibited. The physical exercise should be moderate in amount and of the gentlest sort, and if the patient's occupation tends to stimulate the heart, it must be immediately abandoned. A mild saline purge (ʒij to ʒss—8.0 to 16.0—of Rochelle salts once daily) is quite beneficial.

For the relief of the symptoms referring to the head (tinnitus aurium, vertigo, fulness) and to the precordia (weight and discomfort) arterial relaxants are the best, particularly when arterio-sclerosis is causing cardiac overstrain. Among them nitroglycerin in full doses and veratrum viride are most useful, though the efficacy of both may often be enhanced by the addition of the bromids. In cases of nervous origin the bromids, with preparations of valerian, are the most valuable agents. Nothing, however, is of higher importance than the determination and removal of the cause when possible. After compensation has failed the further treatment is identical with that of cardiac dilatation.



## DILATATION OF THE HEART.

**Definition.**—By dilatation of the heart is meant an enlargement of its various cavities. The walls of the chambers may in consequence be thinner than in health, but much more commonly they are thicker, as in *dilatation with hypertrophy*. Both hypertrophy and dilatation are relative terms, but the latter has reference to that condition in which the cavities are distended out of proportion to the diameter of their walls.

**Varieties.**—(1) *Dilatation with Hypertrophy.*—Here there is a progressive increase in the capacity of the chambers until they attain to large dimensions. The cardiac walls continue of abnormal thickness, yet the vigor of the divisions affected may be relatively diminished to a remarkable degree, owing to the weakening influence of the degenerative processes that attack the hypertrophied muscles. In eccentric hypertrophy the heart-cavities are dilated, but the hypertrophied cardiac walls are sufficiently vigorous to meet the demands of the circulation. This condition should not be regarded as identical with *dilatation with hypertrophy*, but frequently merges into the latter, the size of the cavities now being proportionately greater than is the thickness or the functional power of their walls.

(2) *Dilatation with Thinning of the Heart-walls.*—The diminution in the diameter of the cardiac muscles may be slight if the capacity of the chambers involved be only moderately increased. Instances of this sort are sometimes seen to follow prolonged fever (typhoid). On the other hand, the process of attenuation may reach a high grade, the greatly thinned cardiac wall being scarcely capable of holding the weight of the contained blood.

(3) *Dilatation with little or no variation from the normal cardiac wall* has also been described by some authors. It is to be observed, however, that stretching of a cavity whose walls are normal must be attended with thinning of those walls, unless there has been pre-existing hypertrophy.

**Pathology.**—Dilatation with hypertrophy is generally secondary to valve-lesions, and affects more than one cavity as a rule. It may happen, as in advanced aortic regurgitation, that all the divisions are dilated. The right ventricle is somewhat more frequently dilated than the left, however, for reasons previously adduced. The auricles (especially the left) are more frequently expanded than the ventricles; hence of all the chambers the left ventricle is least apt to dilate. The extent of the relative increase in the capacity of the cavities is variable, and often remarkable. As an example of extreme dilatation of a chamber, the left auricle in cases of mitral stenosis may be singled out; I have seen an instance in which this auricle was capable of containing twenty-two ounces of blood. The septum may be seen to bulge when one ventricle only is stretched. Extensive dilatation of the chambers produces a dilated condition of the auriculo-ventricular rings, which in turn gives rise to relative incompetency. Other cardiac orifices are found to be similarly dilated. Dombrowski<sup>1</sup> has drawn attention to the fact, first pointed out by Wolf, that the surface of the mitral leaflets greatly exceeds the orifice, and Kirschner and Garcin contend that the anterior

<sup>1</sup> "Functional Insufficiency of the Valves of the Left Heart," *Revue de Médecine*, Sept. 10, 1893.



flap alone suffices to close the mitral orifice, "even when the left heart is considerably dilated." Dombrowski believes that functional incompetency is due, in many cases, "to muscular dilatation, producing a separation of the insertions of the papillary muscles, which in systole cannot approach each other near enough to allow the valves to close, the contraction of the papillary muscles only increasing the difficulty." Great dilatation of the left auriculo-ventricular ring is, however, probably an important factor in the causation of relative mitral incompetency. The tricuspid valves, being scarcely competent to cover the corresponding orifice normally, are unquestionably incompetent when that orifice is considerably dilated.

The *shape* of the heart is altered according to the seat and extent of the dilatation. When all the cavities are dilated the organ assumes a globular form, while dilatation of the ventricles only produces broadening of the apical region.

*Condition of the Endocardium and Cardiac Muscle.*—The muscular tissue generally exhibits degenerations (fibroid, fatty, or parenchymatous), but in some cases even microscopic changes are entirely wanting. Important as is the part played by the ganglia in maintaining the nutritive integrity of the heart by supplying nervous force, our knowledge of the alterations that may occur in them in this condition is as yet very imperfect. Ott and others have, however, found them to be degenerated. Opacity and patchy roughening of the endocardium are common.

**Etiology.**—Entering into the causation of cardiac dilatation, there are two essential factors: (1) increased endocardial tension; (2) diminished resistance, due to weakened cardiac walls. Each of these may be the sole cause, though more often they act together.

(1) **Increased Endocardial Tension.**—It is to be premised that a primary and a secondary form occur, the latter being of greater importance clinically than the former. Primary dilatation occurs from a recent obstruction to the circulation of considerable magnitude and at any point throughout the blood-vessel system. A good example is afforded by aortic constriction, in which condition the obstruction of the aortic ring engenders dilatation of the left ventricle by raising the intraventricular pressure; this is, however, quickly overcome by compensatory hypertrophy. In the vast majority of these instances, after a long interval of perfect compensation, a nutritive break-down takes place, with ensuing secondary dilatation.

Other causes of augmented endocardial pressure have been considered in the discussion of Hypertrophy and Chronic Valvular Lesions. In eccentric hypertrophy both dilatation and hypertrophy go hand in hand, until finally the cardiac nutritive functions fail and dilatation at once predominates (dilatation with hypertrophy). Compensation has now been ruptured. Among the exciting factors that may precipitate this accident may be briefly stated—recurrent endocarditis, intercurrent febrile affections which over-stimulate the heart and tend to impair its muscular tissue, general disturbances of nutrition, and, lastly, physical and mental overstrain.

*Acute primary dilatation* may be brought about by sudden, great exertion, as in ascending mountainous elevations. Under these circumstances the heart palpitates violently, there is epigastric pulsation, and

often pain in the cardiac region—evidences of dilatation of the right ventricle. Although in these cases the heart's reserve capacity for work has been exceeded, rest and then quite moderate exercise often restore the conditions to the normal. I have seen acute primary dilatation produced by strong emotion; in such cases sudden contraction of the peripheral vessels occurs, attended with arrest of the heart's action; this soon gives place to violent palpitation and rarely to dilatation. Sudden fright may also, by inducing organic changes, cause acute dilatation.

The remarkable endurance of the athlete and the gymnast is in part owing to the abnormal amount of physiologic cardiac reserve force which they naturally possess, but it is mainly due to the invigorating effect of training. If, however, the training be not so conducted as to symmetrically develop the entire muscular system, or if the exertion be in excess of the reserve functional power of the heart, then acute dilatation may suddenly arise. From this accident recovery may, after a time, take place; sometimes, however, it initiates organic valvular disease, and thus prohibits the further undertaking of unusual feats. The cardiac muscle may be impaired by pericardial adhesions from the extension of fibrous overgrowth to the adjacent myocardium.

*Apparently idiopathic cases* of cardiac dilatation of indeterminate etiology rarely occur.

(2) **Diminished Resistance owing to Weakened Cardiac Walls.**—The occurrences that weaken the cardiac wall are numerous, and not a few lead to acute primary dilatation, such as *myocarditis* due to acute specific fevers (scarlatina, typhoid, typhus). It is especially prone to occur in *rheumatic endocarditis* and *pericarditis*. The *chronic degenerations* (fatty, fibroid) impair the contractile power of the heart. *Nutritional disturbances* of varied origin may induce enfeeblement of the cardiac muscle, such as digestive disorders, ill-ventilation, lack of open-air exercise, and improper or defective food-supply. Dilatation is met with also in *diseases of the blood* (chlorosis, anemia, leukemia).

**Clinical History.**—In acute dilatation the onset is sudden. It is accompanied by such symptoms as dyspnea and cardiac palpitation (both speedily becoming aggravated), and frequently by pain in the precordial region.

The **physical signs** may be incontestable. They are venous pulsation in the neck, a rapid, feeble apex-beat, and a systolic murmur at the tricuspid valves, all of which declare the presence of tricuspid regurgitation. Among signs of subsidiary value are a venous turgescence, a marked epigastric pulsation, and a sudden extension of dulness to the right; the pulse is small, irregular, and exceedingly rapid. In the more chronic form, which arises from slowly-acting causes, or in that which accompanies eccentric hypertrophy or follows simple hypertrophy due to left-sided heart- or lung-trouble, the manifestations that characterize the earliest stage are not at all striking. They indicate weak heart-walls, and such chambers are soon unable to expel their contents during systole. Hence with each subsequent diastole the abnormal amount of blood contained in them is increased. From these facts it is readily seen that the essential causal conditions—increased endocardial pressure and weakened heart-walls—are present and active in facilitating the process of dilatation, once it has commenced. This blood-stasis, as previously pointed



out, often extends from the left heart to the pulmonary vessels, from the latter to the right heart, and finally to the general venous system. Both in the acute and chronic forms, however, *failure of the right ventricle* more often constitutes or determines rupture of compensation and is the harbinger of serious symptoms. Obviously, the symptoms must be those described as belonging to organic diseases of the heart (tricuspid incompetency, in particular).

**Physical Signs.**—*Inspection* in dilatation of the left ventricle shows a diffuse, weak, fluttering, and often a distinctly undulating impulse. The apex-beat will show a greatly diminished vigor in its normal area, or there may be no recognizable area of strongest impulse, as in health. Distinct pulsation in the second left interspace is not rare, and is of ventricular origin. Its feebleness and diffuse character are confirmed by *palpation*. It may be quick and sharp, though always lacking in power. Walsh first made the capital observation—since abundantly corroborated—that the impulse may be visible, yet not palpable. There may be an utter absence of the apex-beat in marked cases. The pulse is small, often rapid, and lacks regularity. *Percussion* shows a lateral increase in dulness to the left, to or even beyond the mid-clavicular line, upward to the second rib, and downward as far as, though rarely below, the sixth interspace, except perhaps, in rare instances, in dilatation with hypertrophy. The lungs in emphysema may to a great extent overlap the heart, with a corresponding diminution in the area of dulness.

*Dilatation of the right ventricle* demands separate consideration so far as the impulse and percussion-dulness are concerned. The normal impulse is largely replaced by the abnormal apex-beat of the right ventricle, which advances to the anterior chest-wall. The chief impulse is now seen and feebly felt, as a rule, below the xiphoid cartilage, or, less commonly, to the right or left of the latter. A wavy pulsation is seen to the left of the sternum, over the fourth, fifth, and sixth interspaces and close to its right edge. If dilatation of the right auricle be associated, as is often the case, a distinct pulsation also occurs in the third right interspace. Dulness reaches to a point 1 inch (2.5 cm.) or more beyond the right sternal border on a level with the fourth interspace. If both ventricles are extensively dilated, dulness extends bilaterally in a transverse direction.

On *auscultation* variable results are obtained according to the state and diameter of the cardiac walls. When thin and not much disorganized, the first sound is much shorter, sharper, and more ringing than in health. In advanced cases the systolic sounds may be feeble and indefinite, and sometimes the first closely resembles the second sound, the long pause being shortened (fetal heart-sounds). The *canter rhythm*, however, is more common. Irregular and intermittent cardiac action are usual phenomena. The abnormal conditions of the two ventricles often differ, or either ventricle may be implicated alone. Hence sounds may differ in intensity. Reduplication may occur, but is not frequent.

Pre-existing organic murmurs obscure the sounds due to dilatation, and, on the other hand, the dilatation may also alter the murmurs (previously audible), and even cause them to disappear, as, for example, in mitral stenosis. Again, dilatation may induce relative incompetency or superadd a murmur, as in cases of chronic valvular disease at the auriculo-



ventricular orifices. It is interesting to recall here that proper treatment may remove a murmur due to relative insufficiency, and that this treatment may, in turn, reproduce an organic murmur.

**Diagnosis.**—This is made readily when there is obtainable a clear history, together with the following characteristic features: a weak, irregular heart-action; an extended, wavy impulse; a small, vigorless, irregular, and intermittent pulse; often an indistinct apex-beat; an outward, upward increase in the percussion-dulness on one or both sides, causing the outline to resemble a square; and a brief, sharp, yet feeble first sound that strikingly resembles the second, which is itself enfeebled.

**Differential Diagnosis.**—*Hypertrophy*, like dilatation, gives rise to an extended area of impulse and of percussion-dulness; hence by the careless observer these conditions are sometimes sadly confounded. From dilatations, where the diagnosis rests upon the points above enumerated, hypertrophy is to be distinguished by symptoms of an opposite nature, such as indicate increased vigor on the part of the heart. The latter are—a slow, heaving impulse; a slow, full, regular pulse; an increase in the area of dulness, chiefly outward and downward; abnormal position of the apex-beat; and the prolonged, dull first and accentuated second sounds. To determine the point at which eccentric hypertrophy ends and dilatation (with hypertrophy) begins is often difficult, but a careful discrimination must be attempted, and I have already discussed the ushering-in symptoms of dilatation following hypertrophy (chiefly of the right ventricle) in connection with Chronic Valvular Disease. Occurring in left ventricular hypertrophy, dilatation first betrays itself by a change in the position of the visible apex-beat and the impulse on palpation. Thus, the maximum point of the apex-beat of hypertrophy very early becomes rounded and indefinite, and later is diffuse and wavy. Its strong, long-drawn, heaving, yet well-defined impulse gives place to the shorter, more sudden shock of commencing dilatation, indicating weakness. These signs, together with a reduction in the strength and an increased frequency or irregularity of the pulse, show the condition to be dilatation with hypertrophy.

The **prognosis** is bad, as a rule, though it may depend upon the causative factors in numerous instances.

**Treatment.**—This in all essential particulars is identical with the treatment of organic heart-affections after rupture of compensation: The etiology in many cases differs from that of the organic valvular affections of the heart, since, next to rest and cardiac stimulants, the removal of the remote and near causes of the dilatation is the most important part of the treatment. Individual cases frequently present special indications, however, and these must be met according to the usual principles, which may be found in appropriate sections of this work. In cases of non-valvular origin digitalis and other heart-stimulants may be omitted, though they should be promptly employed if demanded by a recurrence of the symptoms indicating dilatation. When the dilatation has been overcome, careful attention is to be bestowed upon all the details of the patient's life and sanitary surroundings in order to force his bodily nutrition to the highest point. Every precautionary measure having for its aim the prevention of a recurrence of the dilatation must also be enjoined.

## MYOCARDITIS.

(*Carditis.*)

**Definition.**—An inflammation of the muscle-substance of the heart. It may be acute or chronic.

### ACUTE MYOCARDITIS.

**Pathology and Varieties.**—(1) **Acute Parenchymatous Myocarditis.**

—This is characterized by a granular degeneration of the muscular fibers of the parenchyma of the organ, with a numerical increase in their nuclei. The muscle-structure throughout looks pale, is turbid, and very soft. Many cases of a severe type terminate in fatty degeneration.

(2) **Acute Diffuse Interstitial Myocarditis.**—Here the primary alterations affect the connective tissue of the myocardium; the histologic changes consist in round-cell infiltration.

(3) **Acute Circumscribed Myocarditis.**—In this variety the degenerative processes result in necrosis of the tissues over large or small areas, with abscess-formation. Though usually multiple, these abscesses vary considerably in number, and may rupture either into the various cardiac chambers or into the pericardium. Thus, the purulent contents of the abscess, when there is established a fistulous communication with an endocardial chamber, find their way into the blood-stream and are conveyed to all parts of the arterial system, frequently setting up, here and there, embolic processes of an infectious nature. The blood in turn enters the abscess-cavity, exerting pressure on the walls, and may either produce an acute aneurysmal dilatation of the heart-wall or occasion fatal rupture into the pericardium. More frequently, perhaps, the connective-tissue wall of the abscess yields gradually during the ventricular diastole, when the cardiac aneurysm is formed with corresponding slowness. Occurring in the vicinity of one of the auriculo-ventricular valves, abscesses may cause mitral or tricuspid incompetency. Owing to their tendency to burrow, they may perforate the interventricular septum, thus creating a fistulous connection between the two sides of the heart, and resulting in an intermingling of venous and arterial blood. The abscess may become encysted, then caseous, and finally undergoes a calcareous process.

**Etiology.**—*The causes of myocarditis* are—(a) endo- and pericarditis in the course of rheumatism: it is probable that rheumatic myocarditis may also exist without involvement of the endo- or pericardium; (b) the infectious processes in acute specific fevers; (c) infectious emboli, lodging in the branches of the coronary arteries in connection with septicemia, pyemia, and acute ulcerative endocarditis, and commonly terminating in abscesses (circumscribed myocarditis). The first two of these causes give rise to acute diffuse interstitial and acute parenchymatous myocarditis as a rule. As compared with the female sex, the male suffers much more frequently.

**Symptoms and Diagnosis.**—The symptoms are practically negative. They point to great cardiac enfeeblement, but do not furnish any information beyond exciting a suspicion as to the true nature of the

attack. When cardiac weakness, as shown by a rapid, small, compressible, and irregular pulse, and by attacks of cardiac palpitation and syncope, comes on suddenly in the course of rheumatism, septicemia, or other causal affections, myocarditis may be suspected. Later, signs of venous stasis appear.

The **physical signs** simulate those of dilatation, and may, indeed, be largely dependent upon the presence of the latter condition. Early the action of the heart is tumultuous; the sounds on auscultation are short, sharp, and finally very feeble. Murmurs in myocarditis are not rare, and are not necessarily dependent upon dilatation. Kiehl's work shows the dependence of the valves for their complete closure upon a normal state of the different portions of the heart-muscles, and thus explains these murmurs. Their great variability as to presence or absence is an important point, especially in the diagnosis from murmurs due to endocardial changes. The latter usually coexist with an accentuated pulmonary second sound, while the myocardial murmurs usually do not appear, owing to weakness of the right heart. The special conditions rendering the murmurs audible are great dilatation, softening of the papillary muscle, and abscesses near the valves.

The recognition of *cardiac aneurysm* is made possible by the manner of increase in the percussion-dulness (upward and toward the left) with coextensive pulsation. The symptoms of visceral or cutaneous *embolic processes*, especially when corroborated by the simultaneous development of a murmur and a septic type of fever, should excite strong suspicion of the existence of circumscribed myocarditis.

**Prognosis.**—The diffuse forms are fatal; the circumscribed form may, however, end in recovery. Myocarditis may terminate life suddenly if violent or even active exertion be made.

The **treatment** is identical with that indicated for endocarditis and pericarditis—diseases of which myocarditis is often a complication. The effects of digitalis, particularly when myocarditis supervenes upon old heart-lesions, are quite unsatisfactory. When myocarditis is suspected as an independent condition, absolute rest must be enjoined, the general nutrition energetically maintained, and the more urgent symptoms relieved.

#### CHRONIC MYOCARDITIS.

(*Fibrous Myocarditis.*)

**Definition.**—A gradually developing inflammation of the cardiac interstitial connective tissue, resulting in induration.

**Pathology.**—The characteristic changes may be diffuse, though most frequently they are confined to certain portions of the muscular structure, the left ventricular wall, the septum, and the papillary muscles being the three favorite seats of the process. This is sometimes of antenatal development, and then its usual seat is near the apex of the right ventricle. The hardened spots take the form of more or less rounded patches or broad lines. In color they are gray, grayish-white, or grayish-yellow, the latter tint being due to the intermingling of fibers that have undergone fatty degeneration. Their size is exceedingly variable, some being so minute as to elude detection by the unaided eye, while others measure 1 or 2 inches (2.5–5 cm.) in diameter. Inflamma-



tory induration (contraction) of the *conus arteriosus* of either ventricle causes narrowing of the pulmonary and aortic orifices, with the usual signs and symptoms. Similar changes, by disturbing the functions of the papillary muscles, produce valvular incompetency. Compensatory hypertrophy of the uninvolved portion of the heart is also observed, both the size and weight of the organ thus being increased; the hypertrophic enlargement may frequently be accounted for in part by an associated chronic endocarditis. Sometimes, however, the hypertrophy is occasioned in great measure by general arterial sclerosis. Dilatation of the ventricles follows soon or late, with fresh and grave disturbances of the circulation.

Chronic inflammation usually attacks early the intima of the coronary arteries, and leads to thrombosis, with the formation of anemic infarcts that subsequently undergo sclerotic changes in the muscle-structure. It is probable that most cases of localized fibrous myocarditis have their origin in an obliterating endarteritis. The calloused zone may yield to the endocardial blood-tension, and thus slowly produce saccular dilatation (aneurysm). Microscopically, the affection is characterized by hyperplasia of the interfibrillar connective tissue with subsequent development of new fibrous tissue. Fatty degeneration and atrophy of the muscle-fibers (the latter in consequence of compression by the fibroid degenerated tissue) are also observed. Fragmentation of the muscle-fibers has also been observed. This occurs as a *postmortem* change, and is due to a softening of the interfibrillar substance (the *état ségmentaire* of Renant).

**Etiology.**—The disease is most commonly traceable to the action of one or more of the following factors: an excess in the use of alcohol or tobacco, lead-poisoning, gout, rheumatism, diabetes, chronic nephritis, malaria, and syphilis. Thus, it may be produced by many infections and chemical irritants, the latter, in most cases, first causing a sclerosis of the coronary arteries, to which the patchy fibroid degeneration is secondary. Some of the causes of acute diffuse interstitial myocarditis may by their more slightly irritant effect (owing to the minuteness of the dose of the specific poison) lead to the subsequent development of the general chronic form (*e. g.* rheumatism). Certain irritants that usually engender localized lesions of chronic myocarditis may also affect, though less frequently, the entire myocardium, such as syphilis, alcohol, and gout. Chronic myocarditis may also arise in consequence of a direct extension of the infective inflammatory processes in chronic endo- and pericarditis. It may also follow injuries of the antero-lateral thoracic region. Sex and age possess a predisposing effect, the disease being more common in males than in females, and after middle life than before that period. The right ventricle is apt to be the seat of chronic myocarditis during fetal life, if at all.

**Symptoms.**—Extensive indurated myocarditis has been met with *postmortem* in numerous instances that have been unattended by perceptible symptoms during life. In many of these cases the presence of compensatory hypertrophy accounts for the absence of any symptoms, and it may, therefore, be inferred that mild grades that fail to manifest themselves must frequently exist. The symptoms when present are, almost without exception, untrustworthy for diagnostic purposes, since

they bear a striking resemblance to those of the organic valvular diseases, minus their more characteristic physical signs. Among the earliest phenomena that point merely to failing heart-power are *dyspnea*, and sometimes also, on exertion, *palpitation* and a *sense of heaviness or constriction* in the precordia. The patient suffers from marked general debility, and becomes fatigued in consequence of the slightest physical exertion. Mental inertia is the rule, and chronic mania may come on and last to the close. Later, more positive disturbances of the circulation gradually arise, and when the breathing becomes more difficult (*cardiac asthma*) signs of venous stasis affecting the liver, gastrointestinal tract, and kidneys, and edema finally appear.

Two symptoms that are frequently manifested, and not without some diagnostic import, remain to be mentioned: (1) *Angina pectoris*, which is attributable to the sclerosed condition of the coronary arteries. It occurs in the form of paroxysms of severe pain in the cardiac area, that shoot into the back and down the left arm, accompanied by great anxiety of mind, an anxious countenance, moderate dyspnea, marked precordial pressure, and a feeling of constraint. It is often followed by some form of arrhythmia. Cases occasionally occur in which recurring paroxysms of angina pectoris, with or without arrhythmia, are the only phenomena of the disease.

(2) *Cardiac Arrhythmia*.—Brachycardia is associated as a rule, there being a reduction in the pulse-rate to 50 or even 40 beats per minute. With this decreased rate intermittency is often combined, and various other forms of disturbed rhythm are also observed, though they are less frequent and less significant. Slowing of the pulse does not, however, prohibit the cardiac palpitation that is especially apt to arise during anginal attacks. Disturbance of the rhythm may, on the other hand, be entirely absent.

The pulse is slow, irregular, and of low tension if cardiac atrophy be present. Should fatty degeneration be conjoined, the pulse will be quickened and irregular, and this effect likewise obtains when the patient escapes sudden death and the usual dilatation supervenes.

Chronic myocarditis may be the sole cause of the pseudo-apoplectic seizures that often terminate life abruptly. Preceding the unexpected attack the patient, usually advanced in life, may have experienced from time to time slight vertigo, syncope, and oppression. These seizures may also be caused by a heavy meal or intense mental or physical exertion, and may consist in a momentary loss of consciousness, paralytic symptoms then being usually absent. At other times they last a number of hours, and are accompanied by paralysis which outlasts the coma, as a rule, by a few hours only. Convulsive twitchings may be present. During the attack cerebral hemorrhage occurs, and may leave the patient hemiplegic. It is highly characteristic of these pseudo-apoplectic seizures that they tend to recur, sometimes at intervals of a few hours for a day or two, but more frequently at longer intervals during many weeks or months.

**Physical Signs.**—The impulse may be feebly heaving (sometimes absent); the apex-beat is displaced downward and to the left, while the dull area is enlarged correspondingly in the same direction. Quite early the heart-sounds may be clear and strong, but subsequently they



become weak and muffled. A contraction of the papillary muscles and of the chordæ tendineæ may cause mitral incompetency with its customary murmur.

With the occurrence of dilatation also comes an apical, systolic murmur (due to relative incompetency), with a gallop rhythm of the heart.

**Differential Diagnosis.**—(1) *Chronic valvular disease* can, as a rule, be eliminated prior to the occurrence of secondary dilatation. During this period murmurs do not occur unless the valvular adnexa (the chordæ and papillary muscles) are affected. In the latter event the secondary alterations in the heart, the symptoms, and whole course of the complaint are the same as in certain chronic valvular lesions.

(2) *Hypertrophy and Dilatation.*—In chronic myocarditis *hypertrophy* does not usually reach as high a grade of development as in the majority of the organic valvular complaints and other causal conditions. But after the occurrence of *dilatation*, following indurated myocarditis, the differential diagnosis between the latter and eccentric hypertrophy is purely conjectural.

(3) *Fatty overgrowth* must be distinguished from fibrous myocarditis, and is met with chiefly in brewers, publicans, and butlers. The disease is also found to be specially related to obesity, and sometimes to over-eating and drinking, combined with indolent habits. These subjects suffer more frequently from bronchitis, emphysema, and nocturnal asthma than patients having chronic myocarditis alone. Slight vertigo is common, but true syncopal attacks are rare, according to my observation. In fatty overgrowth the heart-sounds are weak and decidedly muffled throughout; the pulse is weak, though regular as a rule. Marked obesity, however, often obscures the local signs.

The **prognosis** is grave, chronic myocarditis being a fatal disease. Its course and duration, however, are subject to great variations. Among unfavorable surroundings are certain causal and associated conditions, particularly arterio-sclerosis, chronic interstitial nephritis, and diabetes mellitus. On the other hand, if syphilis has been the cause, hope for temporary improvement, if not for actual cure, may be reasonably entertained.

**Treatment.**—The treatment should be managed according to the considerations pointed out in the treatment of Organic Valvular Disease. Rest of body and mind is imperative. Next to this come the dietetic and hygienic details. Residence in a mild climate in winter and a change to the country or to a moderate elevation in summer are matters of the greatest moment to the welfare of the patient. Those rather frequent cases that present, among other complications, such closely united conditions as arterio-sclerosis, gout, and chronic nephritis sometimes do well while sojourning at certain mineral springs, such as Marienbad, Carlsbad, Kissingen abroad, and Bedford or Saratoga at home. These waters must, however, be cautiously used.

When dilatation arises cardiac stimulants are called for, but must be used with an unusual degree of caution. Strychnin has proved itself to be valuable if perseveringly exhibited, and here, as elsewhere, digitalis deserves a trial; its careless administration, however, may give bad results if the pulse be much retarded or arterial sclerosis coexist. For the angina pectoris morphin, administered hypodermically, is to be pre-



ferred. Recurrences of this distressing symptom may be averted by the cautious use of nitroglycerin, the use of which should, however, be limited to cases that seem to be dependent upon arterial degeneration with high tension. Attacks of syncope are most successfully met by the hypodermic use of the diffusible stimulants (ammonia, ether), and at the same time by putting the patient at rest with the head lowered.

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## DISEASES OF THE CORONARY ARTERIES.

It has previously been noted that in pyemia and allied disorders septic emboli may block the branches of the coronary arteries, causing suppurative infarcts (acute circumscribed myocarditis).

It has also been shown that one of the chief effects of sclerosis affecting the coronary arteries is the production of *chronic myocarditis*. The fact that the sudden blocking of one coronary artery by an embolus causes instant death should also be emphasized. In numerous instances in which death has occurred suddenly either thrombotic or embolic obstruction has been the only discoverable *postmortem* lesion. In others the pathologic evidences of local or general atheroma have coexisted. There is at hand much experimental testimony tending to show that ligation or plugging of the coronary vessels in the lower animals causes arrhythmia or even an abrupt arrest of cardiac action; a partial or even slight reduction in the lumen of the coronary vessels by diminishing the supply of blood to the heart-muscle induces degenerations in the latter. In this connection the anatomic peculiarity of the coronary arteries in that they are end-arteries is to be noted, since it affords a ready interpretation of the notable effects following total or partial occlusion.

The blocking of the terminal branches by emboli or by the more gradual formation of thrombi produces the so-called *anemic necrosis* or *white infarct*—a condition that richly deserves brief description:

**Anemic necrosis** (*anemic infarct*) is met with most frequently in the left ventricle and septum, which receive their blood from the anterior coronary artery. The involved areas are small and circumscribed, and present irregular margins that project slightly above the surface. Rarely the infarct is wedge-shaped. Its color is grayish-white or grayish-red, while the central portion is often distinctly white and firm; less frequently it breaks down into a soft detrital mass (*myomalacia cordis*). When softening does not occur the fibers in the affected area lose their nuclei, becoming first hyaline and subsequently sclerotic. The chief histologic changes are of two sorts: (*a*) the striæ of the muscle-fibers are lost, the latter becoming granular and breaking down; and (*b*) the fibers assume a homogeneous hyaline appearance, the nuclei having disappeared.

The *symptomatic* consequences of the lesions are often obscure and unreliable. Sudden death may take place, and rarely this accident may be due to rupture of the heart. Weak and irregular action of the heart, evidences of embarrassed circulation (especially in the cardio-

pulmonary area, as shown by cough and dyspnea), and finally angina pectoris, are among the chief features observed. Death may ensue in the first attack, but more often the patient survives the first and has repeated subsequent seizures. The paroxysms are presumed to be due to sudden occlusion of a branch of the coronary artery, but it should be stated that occasionally in fatal instances of true angina pectoris a total absence of lesions, including emboli, has been noted.

## DEGENERATIONS OF THE HEART.

(a) **Fatty.**—The term “fatty heart” includes two pathologically distinct affections: (1) Fatty degeneration, in which the cardiac muscle-fibers have been converted into fat; and (2) Fatty overgrowth, in which an abnormal quantity of fat is deposited in and about the heart.

### FATTY DEGENERATION.

**Pathology.**—The condition may be either general or localized. Its most frequent seat is in the left ventricle, the papillary muscles and trabeculæ, first appearing as yellowish spots or stripes beneath the endocardium. The affected portions are light yellow or yellowish-brown (faded leaf) in color, due to an associated brown atrophy; they are also soft and friable, and are easily lacerated. The heart is enlarged, and often decidedly so if the process be general, and its walls lack firmness. The microscope reveals characteristic changes: the striæ and nuclei begin to fade, oil-drops and granules appear in the fibers, and finally the latter are occupied throughout by minute globules.

**Etiology.**—Fatty degeneration has already been mentioned as occurring in both the primary and secondary forms of cardiac hypertrophy. It is found also in association with fatty change in other organs in severe forms of primary and secondary anemias. It is most commonly encountered, however, in the cachectic states produced by such chronic diseases as carcinoma and phthisis, and in the course of acute infectious diseases of intense type, all of which may produce the condition. In poisoning by arsenic and phosphorus and in pernicious anemia it advances to a high grade. The various lesions of the coronary arteries previously considered bear a special causal relation.

**Predisposing causes** are—(a) *age*—it being most common after forty years of age; (b) *sex*—it occurs somewhat more frequently in men than in women, notwithstanding the fact that there are predisposing influences at work in the latter that do not obtain in the male sex, such as childbirth and amenorrhea; and, lastly, (c) whatever may be its apparent etiology, it is invariably preceded by a defective nutritive supply to the muscle-cells: this may be dependent on mechanical causes, such as narrowing of the lumen of the coronary vessels, or upon impairment of the oxygen-carrying power of the blood, as in the anemias.

**Symptoms.**—The disease may exist in an advanced form without

noticeable symptoms, though the conditions under which it is most liable to occur often afford premises for suspicions; only rarely is anything more tangible offered than this. The evidences of cardiac enfeeblement are usually present, but in pernicious anemia the pulse may even be full and regular.

*Dilatation* is apt to supervene early, owing to the weakened state of the heart; and hence it is probable that many of the symptoms that have been ascribed to the fatty change are in reality due to secondary dilatation. Among these are palpitation, dyspnea, a small, irregular, and somewhat quickened pulse, and cool and clammy extremities. The heart-sounds are weak, as a rule, and the action of the heart often irregular; later the physical signs of dilatation are almost invariably present, and, as a rule, are progressively intensified. Sometimes sudden, great physical exertion produces equally sudden dilatation, whereupon a canter rhythm and an apical systolic murmur speedily develop. In most instances, however, the symptoms are more gradually brought to light. Breathlessness on exertion is often a striking feature, and syn-copal attacks are sometimes troublesome. The pulse, in consequence of irritation of the inhibitory center in the medulla, often becomes greatly retarded, dropping from the normal rate to 30 or 40 beats per minute, and, in rare cases, to 10 or 12 beats. The fatty *arcus senilis* is devoid of diagnostic value. There are frequent attacks of *cardiac asthma* in the mornings, and these are apt to be accompanied at intervals by *angina pectoris*. Disturbances of the intellect, sometimes taking the form of maniacal delusions, may come on and persist for weeks or even months. Pseudo-apoplectic attacks, such as have been described in connection with Chronic Myocarditis, are also concomitants that point to disturbance of the cerebral circulation. Cheyne-Stokes breathing is among the later manifestations, and I have noticed that these symptoms often occur together, rather than separately, in a given case.

The **diagnosis** is sadly obscure. The history (of the utmost importance), the age of the patient, and the symptoms of cardiac weakness and subsequent dilatation, together with retardation of the pulse, apoplectic attacks, and Cheyne-Stokes breathing, in the absence of precedent hypertrophy merely justify a probable diagnosis. With a clear history and the presence of the more significant symptoms, including the signs of dilatation following hypertrophy, fatty changes may be inferred with some degree of assurance, and yet even this state of affairs should not lead to a positive statement of opinion.

The **prognosis** is as varied as the etiology. Death may come quickly, though oftener the end is reached in a gradual manner, the signs and symptoms of advanced dilatation closing the scene.

**Treatment.**—The cause in each individual case should be determined with as much precision as possible, and when ascertained a bold attempt should be made to remove it. This course often places the patient in the most favorable position for the successful treatment of the cardiac condition; and the method embraces many hygienic and dietetic considerations that assist in improving the nutrition of the cardiac tissue—one of the cardinal aims of a proper system of treatment. Anemia in one form or other plays an important rôle in the majority of the cases, and the particular variety present in each instance must deter-



mine the character of the remedies to be employed. In that large category of cases occurring in certain cachexias (cancerous, tuberculous) the following formula has given gratifying results:

R. Acidi arsenosi,	gr. j	(0.0648);
Ferri sulph. exsic.,	gr. xxx	(2.0);
Strychninæ sulph.,	gr. j	(0.0648);
Quininæ sulph.,	ʒj	(4.0);
Papoid,	gr. xxx	(2.0).

M. et ft. capsulæ No. xxx.

Sig. One after meal-time.

A frequent, irregular pulse and other signs of cardiac failure indicate commencing dilatation, and under these circumstances digitalis should be employed in small doses. When used with perseverance it is of the greatest service in many cases of this sort, and in the form of the powder or the aqueous extract it may be conveniently combined with the above prescription. For the treatment of the more serious evidences of failure of the circulation the reader is referred to the discussion of the organic valvular diseases.

I believe that gentle indulgence in physical exercise and light gymnastics is beneficial, since it tends to invigorate the heart-muscle; it is to be increased in proportion to the manifest improvement in the patient's condition. It sometimes happens, however, that even gentle exercise is badly borne, and it should then be discontinued. I have been in the habit of advising daily inhalations of oxygen gas in this class of cases with good results. Recourse to massage is also in the line of sound practice, but the sittings should not exceed half an hour in duration at the start. The more prominent symptoms may require special measures. The syncopal and anginal attacks are to be handled in the manner indicated for the same symptoms in chronic myocarditis. For the pseudo-apoplectic attacks rest in the recumbent posture, with the head slightly elevated, is useful. Therapeutic agents, as digitalis, ammonia, and ether, may be used hypodermically to stimulate the heart; it is also good practice to withdraw from 12 to 24 ounces (355.0–710.0) of blood directly from a vein. If the arteries be hard and tense, nitroglycerin is of service.

A strictly horizontal posture and the application of ice to the precordial region often quickly terminate the attacks of cardiac asthma, and spartein sulphate, with nitroglycerin, is worthy of a trial. Hot toddy and other diffusible stimulants are valuable adjuvants. Should these remedies fail, hypodermic treatment by morphin is then to be adopted.

#### FATTY OVERGROWTH.

**Pathology.**—The characteristic change consists in a marked increase in the normal fat, particularly in the auriculo-ventricular furrows. This over-production of fat takes place to a greater or lesser extent in every obese person, and may become so excessive as to form a complete enveloping mantle measuring an inch or more in thickness. In these extreme grades the muscular fibers of the organ may, from too great pressure, undergo atrophy and thus become weakened. Dilatation











